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THE TREATMENT OF ACID AND ALKALI BURNS

AN EXPERIMENTAL STUDY

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ALTHOUGH acid and alkali burns of the skin are of frequent occurrence, the treatment of such lesions has not received the critical attention which it merits. A few special types of chemical burns of the eyes have been investigated, but in this situation the caustic is acting under quite different conditions than upon skin. Unlike the eye, skin is protected by a resistant horny layer of cells whose vulnerability is further lessened by its normal oily secretion. A chemical burn may occur only after this protective mechanism has been penetrated either by a strong caustic or by prolonged contact with a less concentrated one.

FIG. 1.—Showing rat 24 hours after exposure for 15 seconds to 70–71 per cent. nitric acid which was treated by washing with water. The control animals and the rats treated by neutralization succumbed in less than 14 hours.

Acids and alkalies produce changes in the tissues which are similar to those caused by heat, but the problem of management is an entirely different one. Heat burns are more or less self-limited in depth because tissue is a poor conductor of heat and any hot material which may splash on skin soon chills. Such a lesion is then in condition to proceed with the normal reparative processes. In acid and alkali burns the destructive action of the caustic may be progressive because any considerable excess of either H or OH ions is incompatible with cell life.[1] The problem of management of such lesions is then primarily one of disposing of the irritant which is still present so that the process of healing may begin.

The principle of neutralization has been the basis of treatment of such burns. Acetic acid has generally been the agent used in alkali burns and sodium bicarbonate in acid burns. Smith[2] and Holland,[3] however, have advocated the use of water as a first-aid treatment and secondary neutralization. This difference of opinion from generally accepted practice was not supported by any observations of burns treated by the two methods under controlled conditions. Because of the fundamental importance of the disposition of the acid or alkali, it seemed desirable to determine the efficacy

of neutralization in contradistinction to simple dilution in experimentally produced burns of this origin.

Classification.—The degrees of cutaneous irritation seen in acid and alkali burns are identical to the changes produced by heat burns of increasing severity. A first degree burn is shown by an arterial and capillary hyperæmia. The dilatation at first involves the superficial vessels, but later progressively involves the deeper subcutaneous tissue partly directly and partly by reflexes. This congestion is accompanied by itching, burning and pain. When the irritant sets up such a vigorous inflammatory reaction that exudate is formed more rapidly than it can be carried away by the lymphatics, vesicles or blebs form, which characterize a second degree burn. Sollman [4] states that this accumulation of fluid takes place between the upper and lower layers of the rete Malpighii. The remaining layers of the rete Malpighii have but little resistance to infection and in this way there may be actual loss of tissue substance. If the corrosive agent continues its action beyond this stage actual cauterization of the tissue or a third degree burn results. This type of lesion is caused by strong acids and alkalies. Such burns show three fairly well-defined areas; first, there is inflammation and hyperæmia at the depth and periphery of the lesion, next a layer of necrotic tissue, and finally a layer in which solution of the individual cells has taken place.

FIG. 2.—Showing rat 24 hours after exposure for 15 seconds to 96 per cent. sulphuric acid, which was treated by neutralization with sodium bicarbonate. (Compare with Fig. 3.)

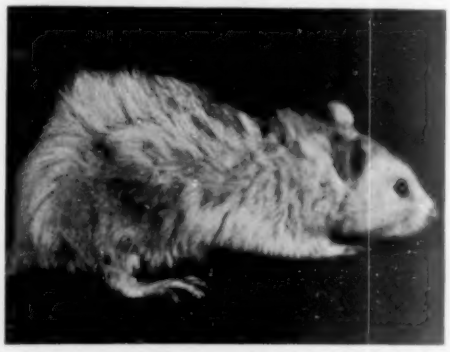

FIG. 3.—Showing rat 24 hours after exposure for 15 seconds to 96 per cent. sulphuric acid, which was treated by vigorous washing with water. (Compare with Fig. 2.)

Method.—Rats were used in all experiments which were done in triplicate. The animals were anæsthetized with ether and a hind leg was immersed for a given period in the test solution, which was at room temperature. Three groups of animals were kept under observation for each concentration of acid or alkali tested. In one group the excess of the caustic was carefully wiped away with cotton and these were considered the controls. The irritant was neutralized in the rats of the second group with five per cent. sodium bicarbonate for the acid burns and one per cent. acetic acid for the alkali burns. The rats in the third group were treated by vigorous washing with water. This was accomplished either by holding the affected part under a running tap or by

placing the animal in a large tank of water. The animals were then placed in separate cages and nothing further was done to alter the course of the tissue reaction to the caustic. If death did not occur promptly in those rats which showed extensive sloughing the animal was sacrificed.

The latent period of sensory stimulation of various concentrations of the same acids and alkalies were determined in three individuals by applying a drop of the caustic upon the inner surface of the forearm. All observations were done in duplicate and the reaction time was measured with a stop watch.

FIG. 4.—Showing rat 24 hours after a six minute contact with 37 per cent. hydrochloric acid. Control animal in which the excess was carefully wiped away. (Compare with Figs. 5 and 6.)

Acid Burns.—The action of concentrated acids [4] consists of withdrawal of water in the formation of acid albumins, in softening of the connective tissues and epithelium and in special situations in solution of calcareous material. The acid albumins are insoluble in moderately strong, but soluble in concentrated or weak acids. The affinity for water is so strong in the case of concentrated sulphuric acid that not only is water withdrawn from the tissues, but the elements hydrogen and oxygen are split off from their chemical combination with carbon leading to charring.

RESULTS

Mineral Acids, Nitric Acid.—When nitric acid comes in contact with skin or hair it gives the characteristic yellow xanthoproteic reaction. The caustic action of a thirty second exposure to 70 or 71 per cent. nitric acid was observed in three rats. Death occurred promptly and autopsy revealed involvement of skin, subcutaneous tissue and muscle to such a degree that it appeared impossible to arrive at a conclusion about what might be accomplished by treatment.

FIG. 5.—Showing rat 24 hours after a six minute contact with 37 per cent. hydrochloric acid which was treated by neutralization with sodium bicarbonate. (Compare with Figs. 4 and 6.)

Three groups of rats were then exposed to acid of this same concentration for a fifteen second interval. There was practically no latent period before activity began. This was evidenced by xanthoproteic reaction of the hair and skin, prompt œdema and complete disability of the extremity. Death occurred in the control animals in three to six hours and autopsy revealed that the depth of the burn extended down to and involved the underlying muscle.

The second group of rats which were treated by prompt neutralization with sodium bicarbonate differed but little from the control animals. The leg became œdematous promptly, and was held rigid in extension. The hair separated readily from the skin. Such animals succumbed in five to fourteen hours and again autopsy revealed involvement of the underlying muscle in the burn.

The animals in the third group which were vigorously washed in water, showed the characteristic xanthoproteic reaction of the hair and skin, but the hair did not pull out readily. (See Fig. 1.) There was marked œdema of the extremity at the end of twenty-four hours, but unlike the previous animals, the treated extremity was useful. All of these rats survived and although some late sloughing occurred, healing took place without any disability.

A series of experiments were performed using 50 per cent. nitric acid for intervals varying from fifteen to sixty seconds. A group of six rats were used to test the effect of the acid for each time interval, in three rats the acid was neutralized and in the others thorough washing was employed. The rats which were exposed to the acid for fifteen and thirty seconds whether treated by neutralization or water, did not reveal a true burn, but there was the usual xanthoproteic reaction. The ones which were immersed for forty-five seconds showed œdema of the extremity and this was equally marked in both groups. The rats which were immersed for sixty seconds and neutralized acted entirely differently, however, from those which were washed. In the former the extremity was curled up, shrunken, dark colored and useless, and death occurred in about eighteen hours. In the latter the only evidence of contact with the caustic was œdema of the foot and characteristic coloration of the hair. None of the animals revealed any evidence of disability and all recovered.

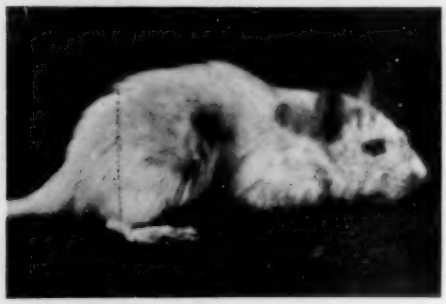

FIG. 6.—Showing rat 24 hours after a six minute contact with 37 per cent. hydrochloric acid which was treated by washing with water. (Compare with Figs. 4 and 5.)

Sulphuric Acid.—Similar experiments were performed with 96 per cent. sulphuric acid. The period of immersion was fifteen seconds. The action upon the skin was so vigorous in the control animals during the next few minutes after the excess had been carefully wiped away that it was deemed advisable to wash after this interval to prevent prompt death. There was immediate œdema, redness, and complete loss of function of the extremity. Death occurred in less than ten hours and in each instance autopsy revealed involvement of muscle in the burn. A group of rats with similarly produced lesions were treated by prompt neutralization with a 5 per cent. solution of sodium carbonate. This procedure caused a tremendous sensory stimulation in those animals in which the anæsthesia was gradually wearing off and a definite increase in temperature of the solution was noted. Death generally occurred in animals so treated in ten hours and invariably in less than twenty-four hours. A third group were treated by neutralization with 5 per cent. sodium bicarbonate and again similar sensory stimulation was seen during the process, but not as great heat evolution was observed. Œdema and redness developed promptly and there was complete disability of the extremity which was drawn up under the animal. (See Fig. 2.) Eighteen hours later extensive sloughing of the skin over the thigh began and death occurred in twenty-four to forty-eight hours. A fourth group was treated by vigorous washing with water. The sensory stimulation which was seen during

FIG. 7.—Showing rat 18 hours after a six minute contact with 50 per cent. sodium hydroxide. Control animal in which the excess was carefully sponged away with cotton. Gangrene of hind leg is evident. (Compare with Figs. 8 and 9.)

the process of neutralization was not observed. There was prompt redness and œdema of the extremity, but this subsided in about twenty-four hours. The leg remained useful and at no time was there any disability caused by the irritant. (See Fig. 3.) In no instance did any late sloughing occur and all animals recovered.

Observations were then made using 50 per cent. sulphuric acid for an interval of thirty seconds. In the control animals a burn occurred which extended down to muscle and the skin of the thigh formed one large slough. No charring occurred. These animals succumbed in about eight hours. Neither the rats which were treated by washing nor those treated by neutralization showed any evidence of a burn.

FIG. 8.—Showing rat 24 hours after a six minute contact with 50 per cent. sodium hydroxide which was treated by neutralization with acetic acid. (Compare with Figs. 7 and 8.)

The effect of immersion for one minute in a 25 per cent. solution was next determined. The controls which were sponged revealed only a slight redness of the extremity but no actual burn. The treated animals revealed no signs of the effect of the irritant. Immersion for one and one-half minutes in a 10 per cent. solution did not produce any lesion even in the control animals.

Hydrochloric Acid.—The caustic action of hydrochloric acid was much less vigorous than that of the acids just described. It was necessary to immerse the extremity in 37 per cent. acid for one minute and allow further contact for five minutes before proceeding with treatment to produce analogous lesions to those previously described. The control animals showed a gradually developing œdema and the leg slowly went into a position of contracture. (See Fig. 4.) The toes became gangrenous and after eighteen hours sloughing of the skin of the thigh developed. Death occurred in about twenty-four hours. The rats which were treated by neutralization showed sloughing of the skin of the thigh at the end of twenty-four hours the leg was useless and death generally occurred in about thirty-six hours. (See Fig. 5.) The animals which were treated by vigorous washing showed some œdema of the extremity and redness of the feet. (See Fig. 6.) At the end of forty-eight hours the hair began to separate, but no sloughing of the skin occurred and all rats so treated survived. An effort was made to produce burns with less concentrated acid, but this was ineffectual because of difficulty experienced with the anæsthetic and protecting the lungs from the fuming acid.

FIG. 9.—Showing rat 24 hours after a six minute contact with 50 per cent. sodium hydroxide which was treated by washing vigorously with water. (Compare with Figs. 7 and 8.)

Organic Acids, Acetic Acid.—It was deemed advisable to determine whether organic acids acted similarly to the mineral acids. The animals were immersed in 99 per cent. acetic acid for thirty seconds. In the control animals the extremity invariably turned an intense red color, but the hair did not show any alteration. All of the rats succumbed in less than two hours and examination revealed that the skin which appeared intact upon inspection was completely destroyed and readily separated from the muscle. Little difference was noted between the animals which were treated by neutralization from those

which were washed. In each instance there was œdema and redness but no actual sloughing of the skin. Exposure to the same concentration for one minute merely increased the severity of the local reaction but no real difference was noted between the rats treated by the two methods. When rats were immersed for one minute and an interval of five minutes was allowed before proceeding with treatment all died regardless of what was done. However, when the interval was lessened to two minutes before proceeding with the treatment, all of the animals from which the acid was removed by washing survived, while two out of three which were treated by neutralization succumbed in less than eighteen hours.

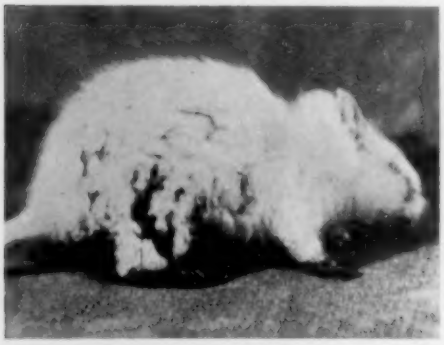

FIG. 10.—Showing rat 18 hours after a six minute contact with 50 per cent. potassium hydroxide. Control animal in which the excess was carefully sponged away with cotton. Gangrene of hind leg present. (Compare with Figs. 11 and 12.)

Trichloracetic Acid.—The caustic action of a saturated solution of trichloracetic acid was observed in three groups of rats. The period of immersion was one minute. The exposed extremity in the control animals immediately became rigid and was held in extension. The skin became milky white within a few minutes, the hair showed no change. Death occurred in such animals in about one and one-half hours. The animals which were treated by neutralization and by washing showed essentially the same picture. The extremity was useless, it was held rigidly in extension and the skin was white in color. The entire leg in all these rats appeared devitalized. Death occurred in the treated animals in ten to twelve hours. Experiments were then done using a half saturated solution and exposing the animals for thirty seconds. The control animals again showed complete disability of the exposed extremity. The rats appeared quite toxic within half an hour after contact with the acid and succumbed in four to five hours. The rats whose lesions were treated by neutralization showed the same characteristic white skin. The leg was pulled up under the body but was used occasionally. At the end of eighteen hours there was moderate œdema and the rats died in twenty-four to thirty-six hours, but at autopsy there was little gross evidence of a burn. The rats which were treated by washing showed œdema of the foot at the end of eighteen hours, but no disability resulted from the lesion and the general activity of the animals did not appear decreased. One rat died fifty hours after exposure to the acid. Another showed the late development of a slough on the lower leg, but the toes remained intact and revealed no deformity.

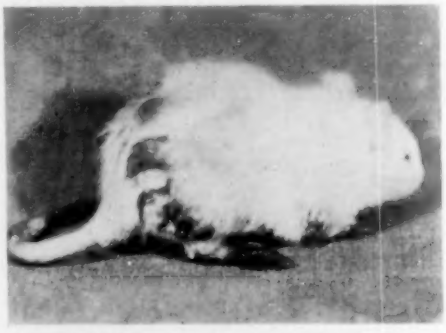

FIG. 11.—Showing rat 24 hours after a six minute contact with 50 per cent. potassium hydroxide, which was treated by neutralization with acetic acid. (Compare with Figs. 10 and 12.)

Alkali Burns.—According to Holland [3] the effect of alkalies is local and limited to the part with which they come in contact. Ammonia, however, may be an exception in view of the three lethal cases reported by Fairbrother,[5] in which death occurred from respiratory and cardiac failure within five hours of the accident. The corrosive action of alkalies are thought to be due to the free alkali combining with the tissue elements forming alkaline albuminates

or with fats to form soaps. Their hygroscopic action of withdrawing water from the cells also contributes to the necrosis. The sloughs which they form are very soluble and consequently alkali penetrates deeply into tissues. It is stated that the total effect upon tissue depends upon the total quantity of OH ions which can be split off under the conditions of the body. The "actual or immediate effective alkalinity of a solution depends upon the dissociated OH ions; but as these combine with the tissues, further OH ions are split off (potential) alkalinity which continue the action.[4]

RESULTS

Sodium Hydroxide.—A long latent period for 50 per cent. sodium hydroxide was observed. It was necessary to immerse the extremity for one minute and allow the contact with the caustic to continue for five additional minutes before proceeding with treatment, in order to obtain a satisfactory lesion. The skin and hair of the control animals varied but little from normal appearance at the end of this six minute interval. Gradually œdema and maceration of the skin with the formation of thick œdematous sloughs developed. (See Fig. 7.) The maceration of the tissue was entirely unlike the destruction seen in acid burns and appeared characteristic of alkali burns. The action of the sodium hydroxide after once begun was rapidly progressive and all of the control animals were sacrificed in eighteen hours. The rats whose lesions were treated by neutralization showed œdema and redness of the foot at the end of twenty-four hours and gradually the skin of the thigh and flank sloughed, however, recovery occurred. (See Fig. 8.) The hair of the rats which were treated by washing remained intact. No sloughing of the skin occurred. (See Fig. 9.) The only evidence of a burn was moderate œdema of the toes and excoriation of the skin of the foot, however, healing took place promptly and without deformity.

FIG. 12.—Showing rat 24 hours after a six minute contact with 50 per cent. potassium hydroxide, which was treated by vigorous washing with water. (Compare with Figs. 10 and 11.)

Potassium Hydroxide.—Here again on account of the long latent period, the same duration of exposure to 50 per cent. potassium hydroxide was necessary as in the experiments with sodium hydroxide. A progressive lesion was again noted in the control animals. (See Fig. 10.) At the end of eighteen hours the foot was swollen and purple in color. The skin was exceedingly moist and mascerated. The toes were shrivelled and gangrenous. The skin was broken in numerous places exposing the muscles of the thigh. The controls were sacrificed at this time. The lesions of the rats treated by neutralization were quite similar. (See Fig. 11.) The extremity was useful but there was great disability. The skin was œdematous and red, and over the thigh sloughing occurred. The toes were dark colored and presented the picture of a dry gangrene. The rats treated by washing, unlike the control and neutralized rats, showed only slight redness of the foot. (See Fig. 12.) There was no œdema and the extremity functioned normally. The hair was everywhere intact and it was difficult to actually demonstrate a burn.

Lime.—An effort was made to produce experimental burns with lime but there are other factors involved than its purely alkaline nature. Calcium oxide may be placed on dry skin and no reaction takes place, the skin likewise tolerates calcium hydroxide, which is not a very active alkali. During the process of slaking, the factor of heat is of tremendous importance. The heat of solution of a single gram of calcium oxide is 18,330

calories. To test the duration of this thermal factor water was added to about 5 grams of calcium oxide. The temperature immediately rose to 98° centigrade. When activity of calcium oxide has once begun on skin there is an addition to this heat factor the avidity of the lime for water which causes further tissue necrosis. Because of this complicated action lime does not lend itself readily for investigation of tissue changes caused by alkalinity.

Sensory Threshold of Skin.—In view of the tremendous difference in the time factor observed in acids and alkalies before tissue injury occurred, it

TABLE I.

Showing Latent Period of Sensory Stimulation of Various Caustics.

Caustic	C. W. M. Latent period	E. C. D. Latent period	W. C. E. Latent period
Acetic Acid			
99 per cent.	14″	11.8″	17″
50 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
25 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
Trichloracetic Acid			
Saturated Solution	5″	14″	13″
Half Saturated Sol.	17″	15″	16″
Potassium Hydroxide			
50 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
25 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
Sodium Hydroxide			
50 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
25 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
Ammonium Hydroxide			
28 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
Nitric Acid			
70 per cent.	4″	2″	8″
50 per cent.	37″	22.6″	45″
25 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
10 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
Sulphuric Acid			
96 per cent.	3.5″	4.6″	4″
50 per cent.	50″	45″	48″
25 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
10 per cent.	3′ No stimulation	3′ No stimulation	3′ No stimulation.
Hydrochloric Acid			
37 per cent.	15″	17″	
25 per cent.	3′ No stimulation	3′ No stimulation	

seemed desirable to determine the latent period upon human skin before a sensory response took place. These observations are summarized in Table I. It is interesting to note that in the highly concentrated mineral and organic acids stimulation occurred in 3.5 to 8 seconds and relatively slight changes in the concentration cause a marked prolongation of the latent period. When a 25 per cent. concentration was reached none of the acids gave a sensory response in three minutes. It is of further interest to note that 50 per cent.

sodium and potassium hydroxide and 28 per cent. ammonium hydroxide gave no stimulation in three minutes.

Discussion.—The results of the experiments with acids and alkalies were quite uniform. In every instance the rats which were washed thoroughly survived longer than those which were treated by neutralization, and the local lesion at any given period after exposure to the caustic revealed less evidence of irritation than those which were treated by neutralization. The striking difference in the results of treatment by the two methods may be due to the additional trauma of heat of neutralization superimposed upon the already existing caustic burn. Heat of dilution must be considered in the rats which were treated by washing, but because of the method used, this heat was promptly carried away, and the sum total of the burn was that due to the caustic agent alone.

The question may be raised whether the results might not be further improved by neutralization of any of the alkali or acid which may still be present after vigorous washing. No experiments to determine this point have been made. It has been noted, however, that dilute acids and alkalies are not very active upon the skin. It is problematic whether the caustic in the slight dilution which is present after energetic washing can cause further tissue injury. It might be added that after such thorough reduction of the concentration that little damage might be anticipated from neutralization of the residual caustic.

CONCLUSIONS

1. Concentrated mineral and organic acids react with skin promptly. As the dilution of the acid increases there is a striking prolongation of the latent period. Concentrated hydrochloric acid is a much less vigorous caustic than either concentrated nitric or sulphuric acids. There is some evidence to suggest that trichloracetic acid is absorbed like phenol and acts as a general protoplasmic poison.

2. Sodium and potassium hydroxide react with skin only after a prolonged latent period.

3. The results obtained in the treatment of experimentally produced alkali and acid burns were decidedly better when the caustic agent was removed by dilution with water than when rendered inert by neutralization.

4. When treatment by neutralization is employed, it should only be used after the maximal amount of the caustic has been removed by thorough washing.

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CONTRIBUTION TO THE STUDY OF BURNS, THEIR CLASSIFICATION AND TREATMENT

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The multiplicity of methods and the difference in opinion in the management of burns shows that we have not yet mastered the question of their treatment. It is because of this that I have made a study of 83 consecutive cases of burns treated at the New York Post-Graduate Hospital. As a result of this study, I desire to call attention to a practical classification and a routine method of treatment that has given gratifying results over a tested period of years.

Classification.—Burns have been classified, as to the agent causing them, into thermal, chemical, electric and radiant energy burns; as to the extent of penetration into the tissues, into: first degree, or erythema forming; second degree, or blister forming; and third degree, or eschar forming. These classifications are explanatory and serviceable from an academic viewpoint, but as the practical trend in surgery is to think of every condition in terms of end-results, the classification of burns rearranged in accordance with this advance is a desideratum.

In observing cases of burns, it has impressed me that a division into two main types—those that will inevitably heal with scar formation and those that will heal without scar formation—immediately puts into the mind of the surgeon a practical classification in which the expected functional end-result is predominant. Thus, under this classification, we recognize a Type 1 or scar forming, and a Type 2, or non-scar forming burn. This classification also, in terms of treatment, tells us that a Type 1 burn will require special treatment to minimize scarring, whereas a Type 2 burn will require little thought to the possibility of scar formation and will necessitate only treatment directed to relieve the inflammation and pain. Under Type 1, is the third degree, and under Type 2, are the first and second degree burns of the old classification. A first degree burn, unless extensive often requires household remedies only, so that this consideration concerns itself really with the second and third degree burns of the old system, which eventually come to the surgeon for treatment.

Further classification of these two types follows a division into three stages based upon the pathological changes going on in the burned area, and treatment is directed to relieve the dominant symptoms in each stage. These stages are not clear-cut. They merge into one another, but for practical purposes they are: the first, inflammatory or dermatitis stage, characterized by an immediate reaction to the causative trauma manifested by all the classi-

cal signs of inflammation; the second, exudative or secretory stage during which the body casts off the dead tissues in the form of a slough; and the third, granulating or reparative stage, during which nature attempts to bridge the loss in continuity of the tissues. Each of these stages has a more or less definite duration time; and I have found them to be 2 to 4 days for the first stage, 6, 10 or more days for the second stage, and weeks for the last stage, depending upon the size and depth of the area burned and the form of treatment applied.

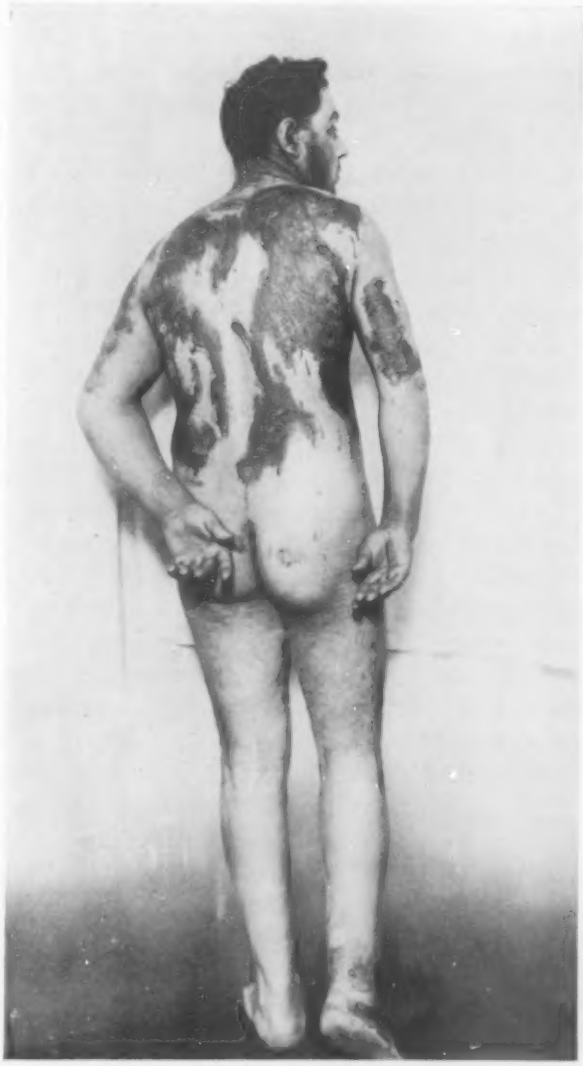

FIG. 1.—Type II burn, ten days after injury.

Pathology.—Pathologically a Type 2, or non-scarring burn varies from a simple hyperæmia of the area without loss of structure to a more severe inflammation and congestion with the early and rapid formation of blisters of various size and extent. The bases of these blebs consist of inflamed skin and the contents of ambre-colored serum, which is fluid in the early stages and gelatinous later. The blood-vessels are congested and a few are thrombosed. The stratum germinativum is markedly swollen and covered by a layer of small leucocytes. In this type of burn, without the superimposition of infection, there is a retrogression of the signs of inflammation with a return to normal of the skin in from ten days to three weeks without leaving a scar. A discoloration or pigmentation, however, may last for a long time. If the burn is of the Type 1, or scar forming variety, there is a destruction of the whole skin layer usually involving the underlying structures. There is a coagulation of the cell contents, destruction or thrombosis

of the blood-vessels of the corium, and the intercellular spaces are filled with broken-down albuminous products. In response to this severe trauma there is a marked inflammatory reaction together with a vasodilatation and alteration in the vascular walls, resulting in an increased permeability of the vessels with consequent loss of circulatory fluids by a pouring out through the vessel walls. This also causes an increased concentration of the blood in extensive burns, and Underhill [1] has called attention to this blood concentration as the result of pathologic exudation which accompanies the vasodilatation.

Healing cannot take place until this dead tissue is removed or allowed to slough away. From this necrotic tissue, due to heat coagulation, there is absorbed into the blood stream a toxin, due very likely to tissue autolysis which is the cause of the toxic or secondary shock. Experimentally, Robertson and Boyd [2] have shown that a substance is produced in burned tissues (in larger quantities in extensive burns of the skin than in those of other tissues) which circulates in the blood, either in or closely adsorbed to, the red blood cells, and which causes the toxic shock and, in some cases, death. Toxic material is produced in increasing amounts following the burning of the tissues until the interval from 24 to 36 hours after the injury. It is produced only as the result of burning living tissues. The toxin is most highly concentrated in solutions of corpuscles suitably treated, and least in the serum. Chemically, this toxin consists of primary and secondary proteoses. Like snake venom, it is made up of two portions, a necro-toxic and a neuro-toxic substance. The former is not destroyed by heat and is diffusible; the latter is thermolabile and colloidal. Robertson and Boyd [3] found no evidence of the formation of antibodies against the burned toxin. Extracts of burned living skin were toxic. Extracts of skin burned post-mortem were inocuous. The contents of burn blisters were not toxic.

Invariably, in all burns of the Type 1 variety, infection supervenes and if no toxic shock is present, the inflammatory reaction becomes aggravated usually after 24 to 72 hours, corresponding to the incubation period of the associated infective organisms; and instead of a simple inflammation, the result of an irritant, we have now to deal with a superficial infected wound and the clinical picture henceforth will be influenced by the extent of the burn and the type and virulence of the infective organism. The organisms we most commonly found were the pus cocci. Because of the frequency of this associated infection, Moorhead [4] aptly defines a burn as "an infected wound due to heat."

Of burns, those produced by moist heat, such as steam or hot liquid, we have found to be the most penetrating, while those produced by flame and electricity we have found to be the more superficial. It has been our experience that the extent of the burned area was of far more importance as to prognosis than its depth. This is in accord with McLeod [5] who further writes as regards prognosis, that a burn of even mild degree may cause a

fatal issue and that this is almost inevitable if the area affected is more than one-third of the total body surface.

Burns by electricity are always associated with flame production so that there is an injury to the deeper tissues by the electric current plus the burn of the skin by the flame. The latter produces a singeing of the skin, as it were, and if the current is not very high we have found the resultant burn readily amenable to treatment. A common site for this type of burn is the face or hands, such as occurs in electric welders and workers on the tracks of electric railways. Schridde[6] has found that burns produced by electricity are histologically identical with those provoked by the application of great heat. Of the commoner gross pathologic findings in death due to burns, degenerative changes in the liver, spleen, kidneys, bone marrow and particularly, the adrenals, were the outstanding findings of Olbrycht[7] and Weiskotten.[8] Recently, Hartman, Rose and Smith[9] have demonstrated an increased output of epinephrin from the suprarenals within a few minutes after burns occur, even in conditions of anæsthesia. The increase may persist for several hours. Under such circumstances, a depletion of epinephrin and of lipoids in the suprarenals may result. The demonstration that burns cause an excessive activity of these glandular structures may help to explain some of the manifestations that have been difficult to interpret. The severe degenerative changes described have been compared to the demonstrated

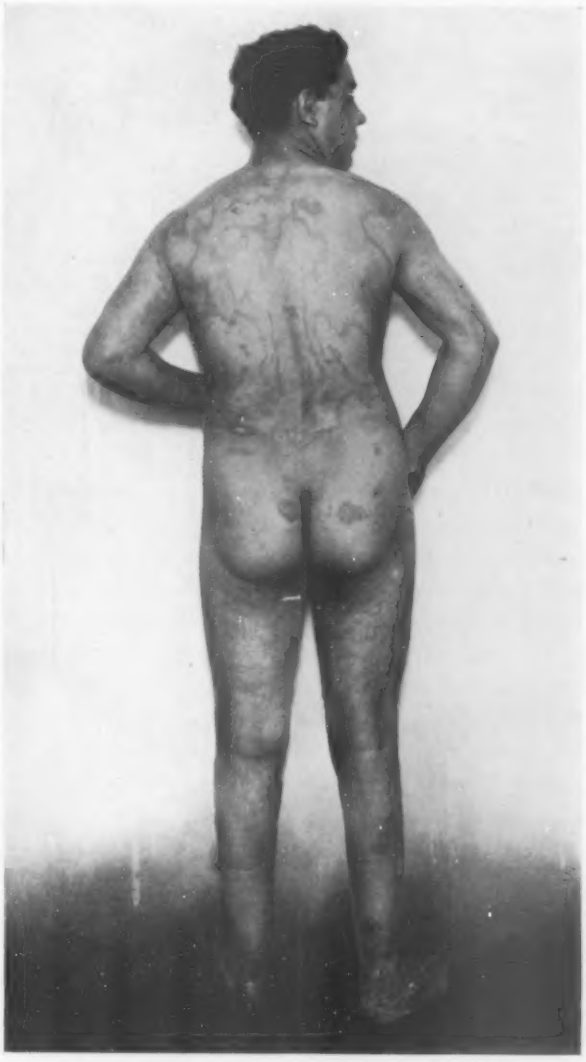

FIG. 2—Same as Fig. 1, nineteen days later, showing pigmentation left after a Type II burn.

occasional comparable effects of diphtheria intoxication. Death, when due to burns, is from traumatic shock when occurring in the first 24 hours, and from toxic shock when occurring in the second to the fourth day. Blumenau [19] reports 43 per cent. of deaths in his series as occurring during this period of toxæmia.

Healing and repair of burns on account of the superficial location is slow, and a large burn may take months to heal unless hastened by skin grafting. The same laws governing repair are active in healing after burns as in wounds made by the scalpel or other instrument, except that the resultant scar, instead of being distributed in the depths of the tissues as in abdominal wounds or deep wounds elsewhere, is spread out on the surface and almost entirely visible. By virtue of this distribution, shrinkage of burn scars is responsible for marked contractures and deformities, and the treatment of a burn should always be with a mind open to this possibility.

Clinical Findings.—Clinically, burns manifest a complex of symptoms referable to the site of the lesion and also as to the general effects. Of the general effects, traumatic shock is the earliest and most important and requires prompt measures to tide the patient over it. It may be a concomitant of either type of burn, if extensive. We have often observed it in a Type 2 burn of the face where the extent and intensity of the burn did not warrant so profound a reaction and we believe the marked reaction following burns in this area to be a nervous manifestation. Bearing in mind the anatomy of the skin with its rich distribution of sensory nerves, we can readily understand that those parts of the body that are nervously unstable will give a more marked reaction. The face, neck and upper chest, being normally the blush area and nervously unstable, are easily influenced by traumatic as well as by other stimuli. Shock occurring after the first 36 hours is toxic in origin. The toxæmia responsible for this may last 2 to 4 days and manifest itself by rise in temperature and pulse rate, drowsiness or restlessness, often vomiting and, in children, convulsions may occur. It is in no way related to the infection that supervenes, but is the result of a toxin liberated by the autolytic process going on in the burned tissues, as explained under the caption of Pathology. Later on, the daily rise in temperature and secondary anemia that follows are due to absorption from a widespread surface infection.

Of the local symptoms, pain is by far the most conspicuous and troublesome, often requiring large doses of morphia for its relief. The urinary findings in our series were mostly negative, with a few cases showing traces of albumin. When appearing, this was a constant finding only during the dermatitis stage, gradually becoming less and less as the burn enters the secretory stage. The clinical picture during this stage is that of a more or less widespread wound infection, secreting a purulent or sero-purulent discharge covered with areas of sloughed tissue, ranging in color from a light yellow to a brown or black. The edges of the wound show a bluish border of epithelium growing toward the centre in an attempt at healing. In the last,

or granulating stage, the appearance is that of a clean granulating wound which may show a tendency toward heaping of the granulations, especially near the centre. There is less of a purulent and more of a serous discharge now and the area continues to grow smaller under the advance of the epithelial covering spreading from the periphery.

Our series presented an increased concentration of the blood, but not as much as reported by others. There was an increase in the nitrogenous waste products, more marked in cases terminating fatally. The chlorides were diminished but not sufficiently to make this a basis of therapy. Marked changes in the liver were evidenced by an increase of bile pigment in the blood and output of urobilin in the urine which, we believe, will prove of importance in therapy.

Total number of cases in this series, 83
- Type 1 variety (scar-forming) .. 16
- Type 2 variety (non-scarring) .. 56
- Type 1 and 2 11

Sites:
- Face 17
- Face and hands 25
- Chest and back 6
- Lower extremities 12
- Upper extremities 17
- Abdomen 2
- More than one-fourth of body... 4

Time elapsed between receipt of burn and hospital admission
- One hour 6
- Less than one day 23
- One day or over 19
- Less than one week 25
- Over one week 4
- Over one month 6

Causative trauma:
- Electricity 47
- Hot liquids 17
- Flame 8
- Gasoline 6
- Steam 4
- Chemicals 1

Age incidence in decades:
- First 8
- Second 4
- Third 29
- Fourth 19
- Fifth 18
- Sixth and over 5

Cases requiring operation 11
- Skin graft 7
- Débridement 4

Complications 7
- Tetanus 3
- Osteomyelitis 4

Deaths 3
- Tetanus 1
- Toxic shock 2

TREATMENT

In approaching the problem of treatment in burns, we considered the physiological functions of the skin which briefly stated are—sensation, excretion, temperature regulation and respiration. We attempted to adopt measures that would not further interfere with the above functions in an already damaged skin. For this reason, we were early advocates of the open air exposure and radiant heat methods of treatment in burns. We dislike to employ any occlusive type of dressing such as the paraffine or ambrine methods, because we believe them to be non-surgical and unnecessary. Since we have adopted the treatment outlined below, we find the necessity for

skin grafting to be less frequent, and in our series of 83 cases, this operation was done only seven times. Further, an occlusive dressing shortly becomes a pus poultice in an actively secreting infected wound tending to retain the secretion, thus promoting the absorption of toxic products.

In our treatment we follow the same subdivision of the stages of burns as outlined under the caption of Pathology, and attention is directed to both the local and general manifestations of the case.

During the first, or toxic stage, shock and toxæmia are the early mani-

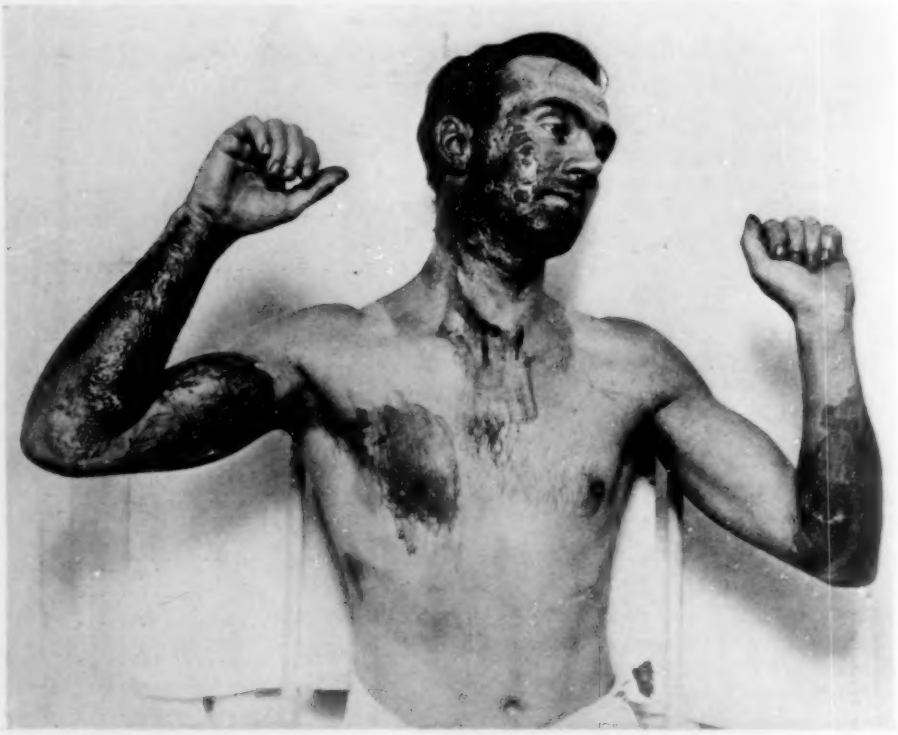

FIG. 3.—Type I and II burn, eight days after injury.

festations. Shock is combated by placing the patient in the "shock position," applying external heat, forcing fluids and relieving pain. External heat may be applied either in the form of radiant heat from electric lights attached to a cage applied over the patient with the lights no nearer than 12 inches from the body. This need not be a highly complicated piece of mechanism and can be readily improvised at the bedside. If electricity is not available, direct heat may be applied by means of hot water bags or automobile inner tubes filled with hot water and placed at the sides of the patient, or hot compresses. Fluids are forced by every avenue of entrance—mouth, rectum, infusion, or hypodermatoclysis. Fluids at this time serve a double purpose—maintaining the circulation and lessening dehydration, and diluting the poisons in the blood stream. Because of the tendency to acidosis, weakly alkaline

fluids are used, and for this purpose, a 5 per cent. sodium bicarbonate solution has given us very good results. The pain associated with the initiating trauma is responsible for this primary shock, and for this reason, sedatives continuously and often in large quantities are indicated. Stimulation for cardiac or respiratory failure are indicated as the necessity for their usage arises.

Locally, the burn is regarded as a wound due to heat, and as such is

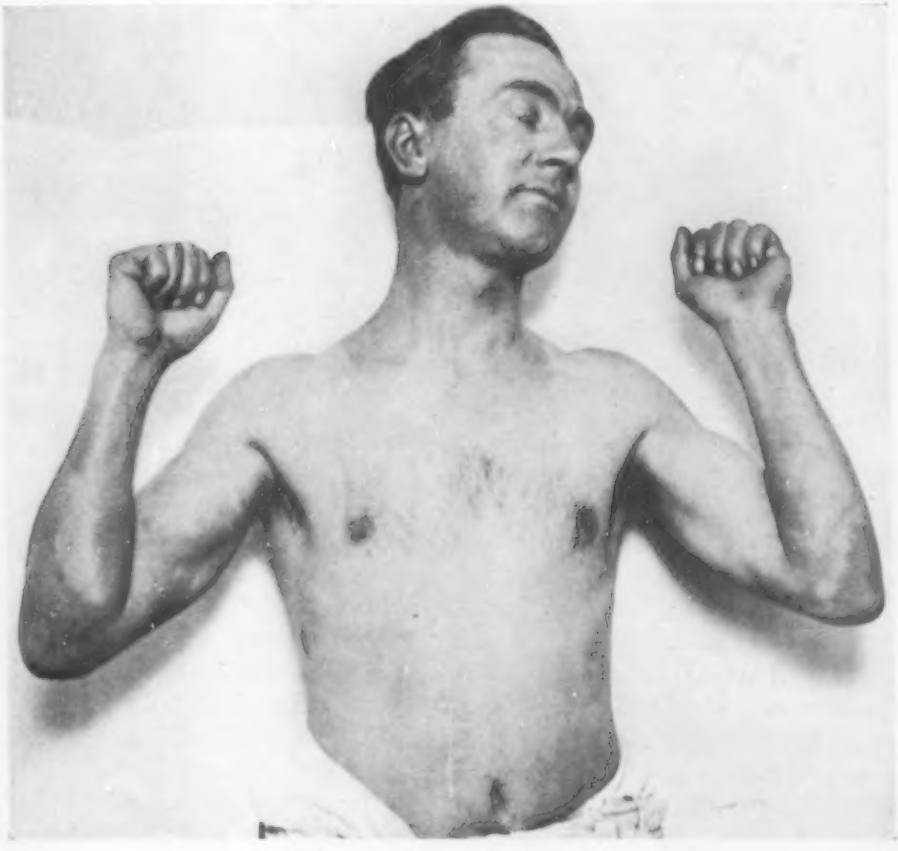

FIG. 4.—Same as Fig. 3, three weeks later.

dealt with surgically as other wounds. Therefore, sterilization with tincture of iodine, one-half strength, is primarily instituted, followed by the application of a wet dressing of a sterile solution of sodium bicarbonate, 10 per cent. This use of iodine seems at first paradoxical, in view of the excruciating pain associated with burns, but its rationale is seen when we remember that the necrotic tissue is insensitive and that the pain is due to the injury and irritation of the nerve endings lying beneath.

The dressing is made of loose fluffs of gauze, in the folds of which several small soft rubber tubes of the Carrel type, or plain tubes with fenestrations in them are incorporated and are allowed to protrude beyond the bandage.

This allows of easy saturation of the dressing periodically without removal of the bandages or the flooding of the bed. This bicarbonate dressing is kept constantly wet and maintained for 24 to 48 hours, by which time the shock, if present at the beginning, will have either entirely disappeared or become greatly ameliorated. The dressing is then completely changed by soaking and a new dressing of similar type is applied and kept on for 2 or 3 days longer, by which time the acute inflammatory character of the wound changes to the milder appearance of the second or secretory stage. At this time, as stated before, the secondary or toxic shock is apt to appear. For this reason, fluids are still pushed. If it should appear, in spite of treatment, blood transfusion will prove of decided benefit. We have had no occasion to use the exsanguination-transfusion treatment, as advocated by Robertson and Boyd[11] and regard it as a heroic form of treatment.

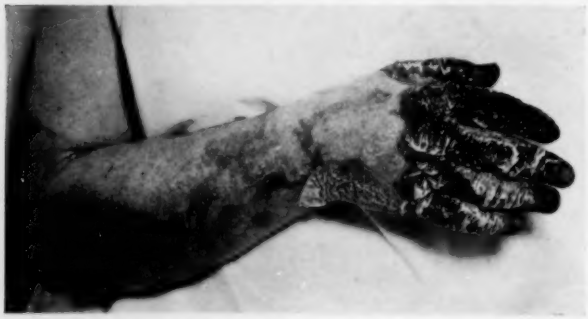

FIG. 5.—Burn of hand, seven days after injury.

All dressings are now removed, blisters are opened aseptically, and the burn is exposed to sunlight during the day. This is done by exposing the wound to the direct sunlight beginning with 5 minutes every 2 hours and increasing to 20 minutes, and in the intervals, to the air. If sunlight is not available, ordinary electric lights may be suspended at a distance of 12 inches from the body and kept there for a period beginning with 10 minutes every 2 hours and increasing to 30 minutes. At night, a wet dressing of sodium bicarbonate solution is applied. Continued use of alkaline dressings is employed to soften the crusts which form because of the air exposure, and to check the process of autolysis going on in the burned tissue. Wiener[12] has shown that the intracellular proteoses act only in a faintly acid medium and that their activity is entirely checked by a slight shift to the alkaline. Where the burn is extensive, we have found sodium bicarbonate baths of great benefit in helping in the separation of sloughs. This is continued until active secretion ceases, which may be from 6 to 10 days.

In children where acidosis is apt to be more marked and where late shock is not an uncommon occurrence, alkaline baths have proven of decided value.

Vogt,[13] Robertson and Boyd[14] and others have demonstrated that the early complete removal of the burned tissues will prevent the development of toxæmia. Others have advised the removal of as much of the necrotic tissue as possible, under a general anæsthetic or large doses of morphine, thus removing the source of the toxæmia. The use of these energetic measures is advocated after the primary period of collapse.

There are cases, however, in which the vitality of the patient might be taxed to such a degree by this therapy that a case with an otherwise favorable prognosis might conceivably end fatally. Further, the idea of the administration of an anæsthetic cannot be accepted too lightly, in view of the fact that there is congestion of the lungs as well as of the abdominal viscera. We have found the use of sodium bicarbonate dressings and baths, if not as heroic, to have proven adequately efficacious at this stage of the burn.

More recently Davidson [15] has been using a 5 per cent. aqueous solution of tannic acid to coagulate the necrotic tissue, and reports good results. He claims that the tannic acid coagulates the injured tissues only, forming a membrane over the burned area. One is not entirely certain, however, whether this tanning process does not tan devitalized tissues which might otherwise have recovered, thus increasing the extent of the slough. Further, the formation of a crust invites infection not only of the aërobic, but particularly of the anaërobic organisms, predisposing to tetanus infection which is not as infrequent in burns as one would suppose, there being 3 cases of tetanus in our series, with one death. Also, the presence of the acid medium would promote the process of autolysis which we aim to arrest. Because of these possibilities, this form of therapy lends itself to selected cases only and cannot be used in the presence of infection.

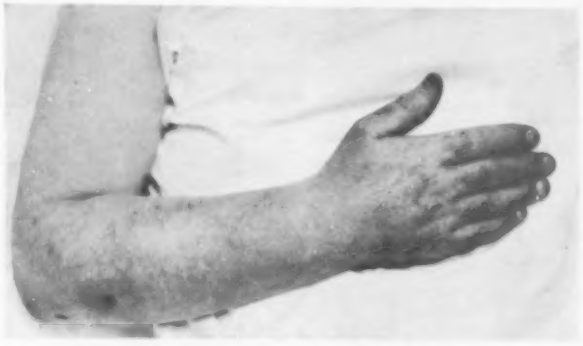

FIG. 6.—Same patient as in Fig. 5, seven weeks later.

After active secretion ceases, which may be from 6 to 10 days, the burned area assumes the characteristics of a granulating wound and the treatment henceforth consists of the sunlight or electric light exposure during the day, and at night a dressing of equal parts of sterile olive and camphorated oil applied on flat pieces of gauze. This dressing, applied at this time, is not painful and the camphor in it acts both as an active stimulant for the growth of granulations and as a germicide. In addition to this, we have been using with gratifying results, exposure to the ultra-violet rays, as follows: After removing all crusts and pus from the surface of the burn, the area is exposed to the air cooled type of lamp, or alpine lamp, at a distance of 24 inches and an exposure time of 3 minutes. The distance is decreased at each treatment, which is every other day, by 4 inches, until 12 inches is reached. The exposure time is then increased—a minute every treatment until 5 minutes are reached, and this is maintained until after epithelization is complete. The effect is a sterilization of the wound, drying up of the secretions, and hastening of granulation and epithelization. To further hasten the

growth of epithelium, we use a dressing of a dram of scarlet red to an ounce of sterile olive oil, and strapping of the periphery of the wound with flamed adhesive plaster.

In cases of Type 1 burn where the absorption from a widespread area over a prolonged period of time produces an exhaustion of the patient, periodic transfusions are of marked benefit and enhance the processes of repair.

During all these stages, the postural position of the patient is of the greatest importance. If the burn is situated near any of the folds or creases of the body, these should be mobilized early, and where immobilization is indicated, on account of excessive pain, it should always be in a position of maximum give, *i.e.*, in complete abduction and extension of the part. Although contractures are spoken of as a late manifestation of burns, it is at this time that they can be prevented, hence the importance of posture as a prophylactic measure.

The treatment outlined above holds true for burns anywhere in the body, except the face. Here the first stage is carried out as above, but when exposure to the sun and light is instituted, the night dressing consists of a 10 per cent. boric acid ointment. For the eyes, 10 per cent. argyrol installations and boric acid lavages are early instituted. To prevent contractures, by means of mobilization of the face, early movements should be encouraged and blowing out the cheeks, wrinkling the forehead and the use of chewing gum have proven helpful.

Skin Grafting.—In following out the above routine of treatment, we have found the use of skin grafting becoming more and more an indication in cases that have had their preliminary treatment elsewhere. The type of graft to be used is largely a matter of individual choice or custom of the surgeon. We have been in the habit of applying the following rule: Where the burn is extensive in area but not in depth, we use the Thiersch type of graft; where the area is small and shallow, the pinch type, and where the burn is deeply penetrating, the flap type of graft. In all cases, wherever possible, auto-graft is desirable. Where this is not possible, a donor of the same blood grouping will give a better chance of "take."

Débridement should be used in selected cases only, not as a routine. In burns, it has very much the same indication as in compound fractures and must be chosen with a good deal of care. It is indicated in Type 1 burns where the slough is localized. If the burn is too extensive, it subjects the patient to too great a shock. In children, it should be done very, very rarely. Débridement, as commonly practiced, is sacrificing of tissues. Skin grafting is repair. Therefore, it is a hospital problem and, indeed, a problem in plastic surgery.

CONCLUSION

1. Classification of burns into scarring and non-scarring types constantly keeps the functional end-result in mind.

2. The knowledge of a Type 1, or scarring burn, will require special

treatment to minimize scar, whereas a Type 2, or non-scarring burn will not require special treatment, makes this classification of practical importance.

3. The open air and radiant light method of treatment have proven adequate and serviceable over a prolonged period of time.

4. It has lessened scar formation and decreased the frequency of skin grafting.

5. It has taken burns from the Carron oil, salve and fertilizer type of management and placed their treatment upon a surgical basis.

6. The open air method of treatment makes the resultant scar more uniform in appearance and more resistant to the rigors of climate.

7. Débridement should be employed only after careful study and consideration of the individual case.

8. There are two types of shock—primary or traumatic, and secondary or absorptive—due to anaphylaxis from protein-split products. Late shock is due to absorption from infection, the organism usually being a coccus.

9. An important part of the treatment is prevention of infection, irregular scars and contractures.

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POST-OPERATIVE WATER METABOLISM AND THE INTRADERMAL SALT SOLUTION TEST*

A PRELIMINARY REPORT

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In the post-operative treatment of patients one of the most important problems is that of the water metabolism of the body. Dean Lewis[1] last spring said, . . . "there is a tendency to administer too great amounts of fluid, and some one should attempt to determine the amounts required in different groups of cases to maintain the water balance." It is a problem, therefore, how much water should be given post-operatively in the various types of cases and what is the most satisfactory method of giving it in the individual case.

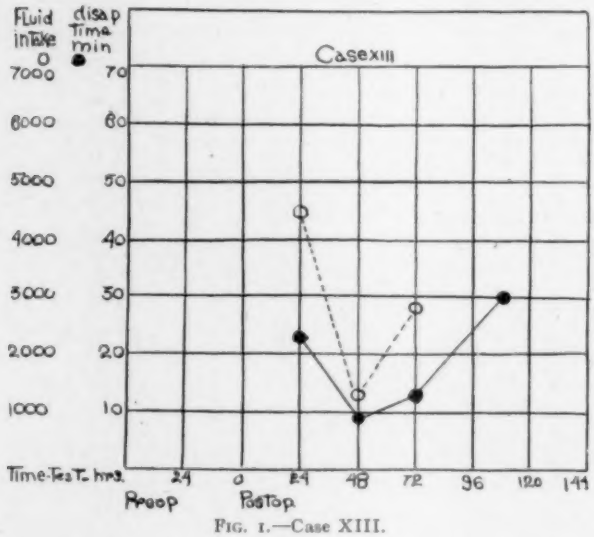

Fig. 1.—Case XIII.

There are a number of tests that are used or can be used in determining the degree of dehydration that exists in the organism. The amount of water that can be absorbed by the various routes is a rough indication of the hydrophilic state of the tissues. The clinical symptom of thirst, of course, is an indication of the need of the body for water. This is grossly unreliable, however, for the correlation between the degree of thirst and the amount of tissue dehydration is not definite, and in the second place the liberal use of morphine masks subjective symptoms. The amount of urine voided is probably the most useful test of the water metabolism. There are objections to it, however. The determination of the hæmoglobin percentage of the blood or the refractorimetric estimation of the plasma proteins are other methods applicable to this problem. It is conceivable, however, that a considerable degree of tissue dehydration can exist before the water content of the blood

* Read before the Philadelphia Academy of Surgery, December 6, 1926.

is altered. A method which seemed to offer the possibility of objective measurement of the need of the tissues themselves for water seemed to present itself in the disappearance time of an intradermally injected quantity of salt solution.

The mechanism of the test is disputed. It is bound up with the problem of œdema in general. Some workers have explained it on the basis of Martin H. Fischer's [2] work, who pointed out that fibrin absorbed more water in an acid solution than in a neutral one, and who offered an explanation of nephritis, œdema, and other pathological processes on these basic facts. There are certain fundamental, experimental objections to this theory. Another theory, that perhaps explains the phenomenon in our type of cases, depends on the balance between capillary pressure and the osmotic pressure of blood.[3, 4]

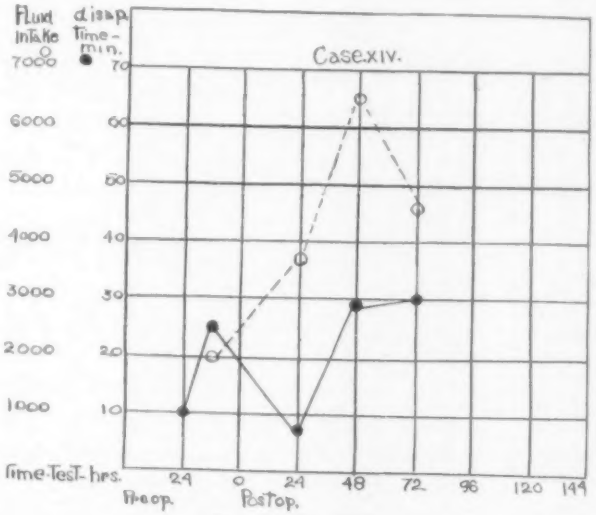

FIG. 2.—Case XIV.

In 1923, McClure and Aldrich [5] first described the intradermal salt solution test. Their method was to inject intradermally 0.2 c.c. of an 0.8 per cent. sodium chloride solution and note the disappearance time of the wheal. They found that the normal disappearance time was roughly sixty minutes, but that in œdematous conditions (nephritis, cardiac decompensation, anæmia) the disappearance time was decreased to as low as three and seven minutes in two cases. Since then, reports by a number of investigators have appeared, applying this test to a variety of conditions. In nephritis [6] it was shown that a reduction in disappearance time preceded the clinical evidence of œdema and that an increase in the reduced disappearance time preceded the disappearance of œdema. In the toxæmias of pregnancy [7] a decreased disappearance time has been demonstrated, and the more reduced the disappearance time the severer the case. Patients with scarlet fever,[8] diphtheria, and pneumonia [9] have been found to have a reduced disappearance time, the reduction being parallel to the severity of the intoxication. The administration of thyroid extract to myxœdematous patients has been shown to decrease the disappearance time of the wheal.[10]

Experimental interference with the peripheral circulation in rabbits was found to decrease the disappearance time.[11] The normal time of disappearance was found to be reduced in such conditions as diabetes with gangrene, endarteritis obliterans, intermittent claudication, and arterial emboli.[12] Read-

ings of ten minutes, for example, were found only in tissues immediately above areas of gangrene, while readings between ten and twenty-five minutes were held to be suggestive of developing gangrene. With varicose veins the rate of disappearance varied according as the member was elevated or dependent. A case was reported [13] in which it was difficult to tell whether the condition of the patient was due to shock or hemorrhage after a crushing injury to the thigh. A normal disappearance time of the intradermal salt solution in the wheal seemed to rule out circulatory damage to the member and the condition was held to be due to shock. Autopsy proved there had been no rupture of the vessels. A circulatory disorder masquerading as a traumatic condition was demonstrated by the test. We see, therefore, that although the test has been used in surgical conditions, it has not been used primarily to determine the nature of the water balance of the body.

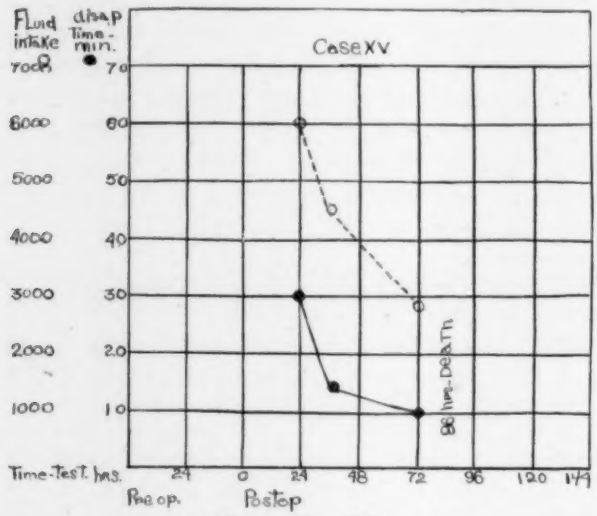

FIG. 3.—Case XV.

The technic employed by us follows: From a tuberculin syringe 0.5 c.c. of a sterile 0.85 per cent. sodium chloride solution is injected intradermally into the skin overlying the peroneal tendons directly above the external malleolus. Two wheals are made about 2 cm. apart and the time required for their disappearance to light touch "unassisted by inspection" is determined. We found that, in a small series of normals with our method of injecting only 0.05 c.c., the disappearance time was about thirty minutes—although occasionally some lasted as long as fifty or sixty minutes. Below

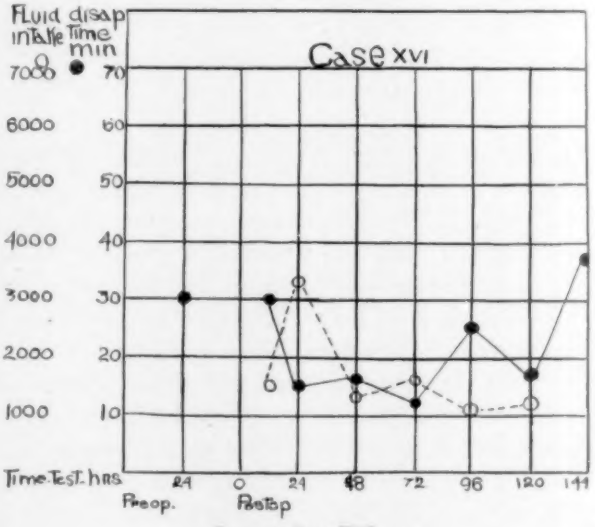

FIG. 4.—Case XVI.

twenty-five to thirty minutes we felt was abnormal, and anything below ten minutes was distinctly abnormal. The reason we elected to use such a small wheal was because of the time-saving factor on a busy surgical service. Our normal figure, therefore, of thirty minutes is much shorter than that used by previous observers. It is possible, too, that the end point of a smaller wheal is less satisfactory than a larger one. In interpreting the disappearance of the wheal, one must bear in mind the other conditions beside dehydration in which there is a decrease in the wheal disappearance time, *viz.*, circulatory disturbances (such as occur in emboli, thrombosis, arteriosclerosis, thromboangeitis obliterans, and diabetes); conditions producing œdema (cardiac and renal disease); toxic conditions (pneumonia, scarlet fever, diphtheria, toxæmias of pregnancy), and anæmias. The chloride metabolism in intestinal obstruction may be an additional factor.

The small number of observations that follow are offered merely as suggestions. We are by no means sure of the mechanism involved in this test or its ultimate value as a test for body dehydration.

Case I.—F. F., male, age sixty-eight years. Symptoms of repeated hæmatemesis for several days. Diagnosis: Duodenal ulcer. X-ray uncertain. Unoperated. Disappearance time twenty minutes.

Case II.—M. M., male, age twenty-four years. Diagnosis: Right inguinal hernia.

Time of Test	Wheal Disappearance Time
Pre-operative	30 + minutes
Post-operative, 24 hrs.	35 + minutes

Case III.—R. G., male, age twenty-six years. Diagnosis: Right inguinal hernia.

Time of Test	Wheal Disappearance Time
Pre-operative	30 + minutes
Post-operative, 24 hrs.	35 + minutes

Case IV.—G. G., male, age thirty-five years. Diagnosis: Duodenal ulcer; chronic appendicitis. Operation: Oversuture of ulcer; posterior gastro-enterostomy; appendectomy.

Time of Test	Wheal Disappearance Time	Intake	
Pre-operative	32 minutes	Hypodermoclysis	Enteroclysis
Post-operative, 35 hrs.	30 minutes	3250 c.c.	4000 c.c.

Case V.—E. L., female, age fifty years. Diagnosis: Carcinoma of stomach. Operation: Subtotal gastrectomy. Posterior Polya.

Time of Test	Wheal Disappearance Time	Intake	
Pre-operative	45 minutes	Hypodermoclysis	Enteroclysis
Post-operative, 34 hrs.	35 minutes	4000 c.c.	3000 c.c.

Case VI.—M. L., female, age fifty-seven years. Diagnosis: Right pyonephrolithiasis. Operation: Right nephrectomy and transfusion.

Time of Test	Wheal Disappearance Time	Intake
Pre-operative	50 minutes	(Patient bled profusely—transfusion, 500 c.c.; intravenous, 500 c.c.
Post-operative, 5 hrs.	13 minutes	
Post-operative, 24 hrs.	15 minutes	Total intake, 3400 c.c.
Post-operative, 13 days	30 + minutes	

CASE VII.—L. M., female, age fifty-seven years. Diagnosis: Pernicious anæmia. Chronic calculous cholecystitis. Operation: Cholecystectomy; splenectomy. Transfusion, Hb., 37 per cent.; red blood-cells, 2,300,000.

Time of Test	Wheal Disappearance Time	Intake
Pre-operative	2 minutes	Blood transfusion, 500 c.c.
Post-transfusion	5 minutes	
Post-operative, 10 hrs.	8 minutes	3000 c.c. (?)
Post-operative, 11 days	4 minutes	1500 c.c. per day (Hb., 51 per cent.; red blood-cells, 2,600,000)

CASE VIII.—V. M., female, age eighteen years. Diagnosis: Post-operative adhesions. Operation: Exploratory laparotomy; lysis of adhesions.

Time of Test	Wheal Disappearance Time	Intake
Pre-operative	?	
Post-operative, 30 min.	50 minutes	
Post-operative, 24 hrs.	50 minutes	2500 enteroclysis
Post-operative, 72 hrs.	25 minutes	650 mouth (24 hrs.)

CASE IX.—H. S., female, age forty-five years. Diagnosis: Acute calculous cholecystitis. Operation: Cholecystectomy.

Time of Test	Wheal Disappearance Time	Intake
Pre-operative	?	
Post-operative, 30 min.	45 minutes	
Post-operative, 24 hrs.	35 minutes	3500 c.c. enteroclysis
Post-operative, 72 hrs.	30 minutes	1250 c.c. by mouth (24 hours)

CASE X.—R. S., female, age thirty-six years. Diagnosis: Chronic appendicitis. Operation: Appendectomy.

Time of Test	Wheal Disappearance Time	Intake
Pre-operative	32 minutes	
Post-operative, 30 hrs.	55 minutes	4500 c.c. enteroclysis
Post-operative, 52 hrs.	75 minutes	210 c.c. (mouth), 2500 enteroclysis
Post-operative, 72 hrs.	55 minutes	(?) intake by mouth

CASE XI.—M. S., female, age nineteen years. Diagnosis: Trophœdema of left foot. Operation: Exploratory laparotomy.

Time of Test	Wheal Disappearance Time		Intake
	Foot	Leg	
Pre-operative	2 min.	18 min.	
Post-operative, 24 hrs.	2½ min.	45 min.	600 c.c. by mouth
Post-operative, 48 hrs.	3½ min.	40 min.	?
Post-operative, 72 hrs.	19 min.	40 min.	?
Post-operative, 96 hrs.	20 min.	20 min.	?

CASE XII.—C. E., male, age sixty-three years. Diagnosis: Carcinoma of stomach. Operation: Exploratory laparotomy.

Time of Test	Wheal Disappearance Time	Intake
Pre-operative	20 minutes	
Post-operative, 6 hrs.	17 minutes	600 c.c. enteroclysis
Post-operative, 24 hrs.	17 minutes	2000 c.c. enteroclysis
Post-operative, 48 hrs.	?	
Post-operative, 72 hrs.	42 minutes	Fluid by mouth (?)
Post-operative, 96 hrs.	30 + minutes	

CASE XIII.—S. K., female, age forty years. Diagnosis: Jaundice, unknown etiology. Operation: Choledochotomy; cholecystostomy.

Time of Test	Wheal Disappearance Time	Intake	
Pre-operative	?	Enteroclysis.	Hypodermoclysis
Post-operative, 24 hrs.	23 minutes	2500 c.c.	2000 c.c.
Post-operative, 48 hrs.	9 minutes	1290 c.c. by mouth	
Post-operative, 72 hrs.	13 minutes	2820 c.c. by mouth	
Post-operative, 108 hrs.	30 minutes	?	

CASE XIV.—M. C., male, age forty-eight years. Diagnosis: Carcinoma of sigmoid. Operation: First stage, Mikulicz.

Time of Test	Wheal Disappearance Time	Intake		
Pre-operative	10 minutes			
Pre-operative, 12 hrs.	25 minutes	2000 c.c. hypodermoclysis		
Post-operative, 24 hrs.	7 minutes	3700 c.c. hypodermoclysis (++ vomiting)		
Post-operative, 48 hrs.	29 minutes	Hypodermoclysis 4300 c.c.	Mouth 1200 c.c.	Intravenous 1000 c.c. (++ vomit)
Post-operative, 72 hrs.	30 minutes	3200 c.c.	900 c.c.	500 c.c.

NOTE.—Directly after intravenous glucose injection patient had a chill and the temperature, pulse, and respiration rose to 104.2, 140, 38, respectively. The wheals disappeared in four minutes, but the following morning disappeared in thirty minutes.

CASE XV.—A. K., female, age thirty-six years. Diagnosis: Carcinoma jejunum. Operation: Entero-enterostomy.

Time of Test	Wheal Disappearance Time	Intake		
Pre-operative	?	Hypodermoclysis	Enteroclysis	Mouth
Post-operative, 24 hrs.	30 minutes	3500 c.c.	2500 c.c.	....
Post-operative, 36 hrs.	14 minutes	4000 c.c.	500 c.c.	60 c.c.
Post-operative, 72 hrs.	10 minutes	2500 c.c.	Transfusion, 350 c.c.	
Post-operative, 86 hrs.	Death			

CASE XVI.—E. Mc., female, age seventeen years. Diagnosis: Hæmolytic icteroanæmia. Operation: Splenectomy.

Time of Test	Wheal Disappearance Time	Intake	
Pre-operative	30 minutes	Enteroclysis	Mouth
Post-operative, 12 hrs.	30 minutes	1500 c.c.	80 c.c.
Post-operative, 24 hrs.	15 minutes	3390 c.c.	?
Post-operative, 48 hrs.	16 minutes	....	1320
Post-operative, 72 hrs.	12 minutes	....	1680
Post-operative, 96 hrs.	25 minutes	....	1080
Post-operative, 120 hrs.	17 minutes	....	1230
Post-operative, 144 hrs.	37 minutes	....	740 ++ (?)

CONCLUSIONS

1. There is frequently a reduced disappearance time of the intradermal salt solution wheal, post-operatively.

2. We have suggested this test as a method for determining the need of the tissues for water post-operatively.

3. This test may be used to show the adequacy of the post-operative introduction of fluids by the various methods.

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CHRONIC STRAIN OF THE LUMBAR SPINE AND SACRO-ILIAC JOINTS*

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Every diagnostician is familiar with the fact that systematic palpation of abdomens reveals deep-seated midline tenderness in a fairly high percentage of cases. This tenderness is usually ascribed to "aortic tenderness," to hypersensitive sympathetic nerves or ganglia or to the dragging of ptosed viscera. None of these theories seems to explain the clinical pictures satisfactorily.

On careful examination of these cases it will be found that the tenderness is sharply limited to the area of the intra-abdominal vertebral bodies and that all aspects of the vertebral bodies (and intervertebral discs) that are accessible to palpation are uniformly tender. The great majority of the patients who exhibit marked midline tenderness also have an isolated area of tenderness in each iliac fossa.

That the midline tenderness might be due to "aortic tenderness" is suggested by the fact that the patient does not experience any discomfort when the examiner places his finger tips on the midline above the umbilicus and exerts barely enough pressure to feel the pulsation of the aorta, but when the examiner then firmly presses his finger tips only a fraction of an inch deeper, the patient will writhe out from under his fingers because of the pain which is produced. That the tenderness is not limited to the aorta, however, is easily demonstrated by the examiner finding that his finger tips (with palm facing the spine), pressed deeply over the outer edge of either rectus muscle into the corresponding paravertebral gutter, do not cause any discomfort, but that when he "hooks" his fingers inward, toward the midline, the patient will manifest distress as soon as and wherever the fingers encounter the resistance of the lateral surfaces of the vertebræ.

The ramifications of the sympathetic nervous system are so very widespread within the abdomen (Fig. 1) that it is inconceivable that any purely sympathetic lesion could be so consistently and sharply localized to the area represented by the vertebral bodies. Furthermore, it is improbable that the sympathetic system is endowed with sensory fibres or terminals that would manifest a sensation of pain on pressure stimuli.

Because of midline tenderness being found so commonly in patients with visceroptosis has led to a rather general acceptance of the theory that it is caused by the ptosed viscera dragging on their attachments to the posterior parieties. But this theory likewise seems untenable, because of the utter improbability of ptosed viscera being able to exert a sufficiently concentrated drag to produce tenderness over every palpable part of the vertebral bodies

* Read before the Philadelphia Academy of Surgery, December 6, 1926.

and yet not produce any tenderness away from the spine itself. Except for the possible factor of intestinal stasis, it is improbable that ptosed viscera

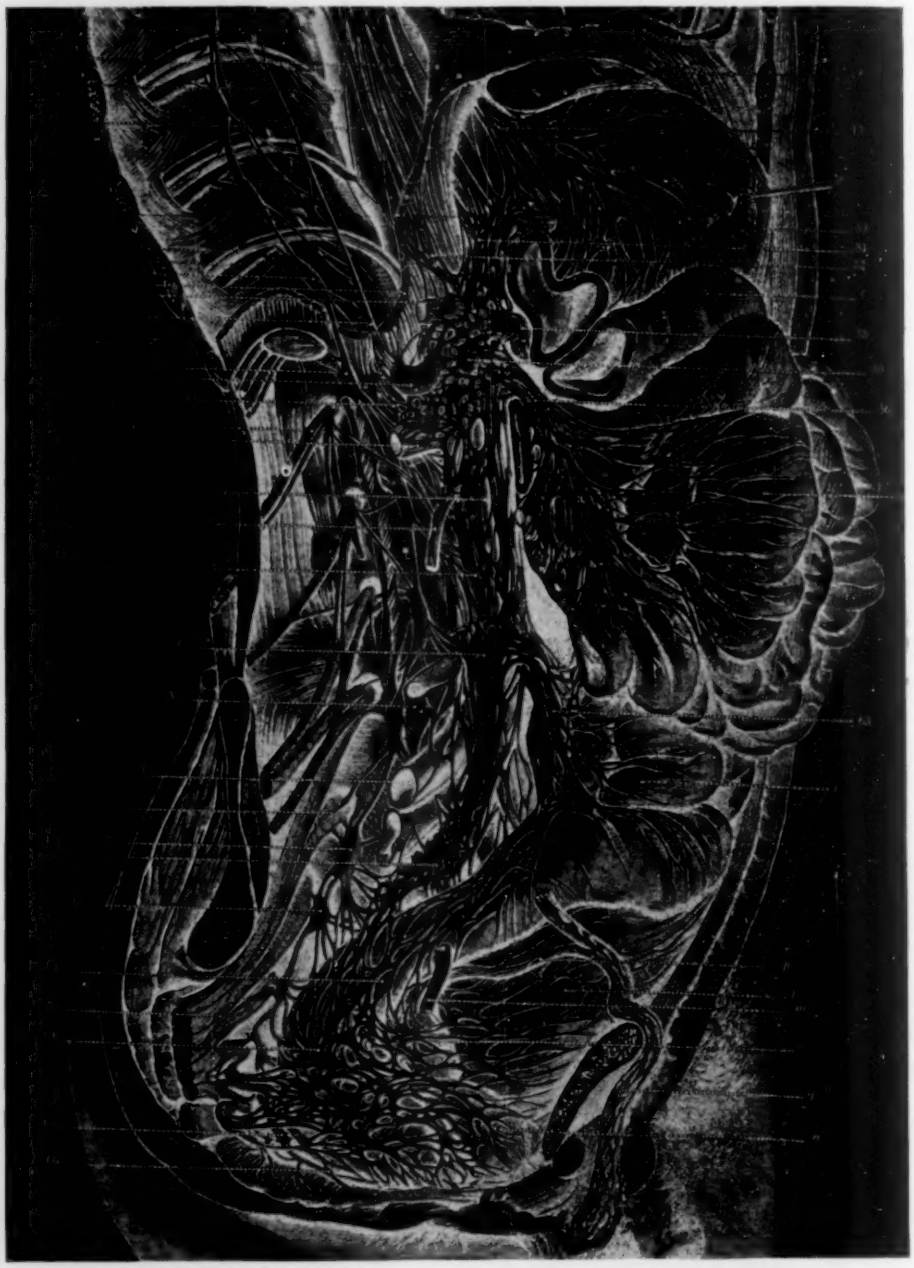

FIG. 1.—The intra-abdominal sympathetic nervous system. (From Broesike's Anatomy.)

exert any more pull than do viscera in normal position. Any increased weight of viscera due to intestinal stasis mainly affects the colon, and especially the

cæcum, and should be manifested chiefly at the site of attachment of the definitive mesocolon. It would therefore seem that any tenderness induced by pull on the definitive ascending or descending mesocolon should be located lateral to the spine and not along a considerable length of the intra-abdominal spine itself.

I am inclined to the belief that chronic midline tenderness is usually due to chronic sprain of the vertebral joints, although it may occasionally result from localized or diffuse arthritis. The great majority of patients who exhibit midline intra-abdominal tenderness are of the asthenic type and have visceroptosis and exaggerated lumbar lordosis. Any increase in the forward curvature of the lumbar spine tends to widen the anterior aspect of the intervertebral spaces. (Figs. 2, 5 and 8.) The more marked the lordosis the greater is the strain on the periarticular structures. This increased postural stress cannot be adequately met by the atonic skeletal musculature, and the ligaments or their periosteal attachments surrounding the front and sides of the vertebral bodies as far down as the top of the sacrum are therefore continuously subjected to undue strain resulting in chronic tenderness.

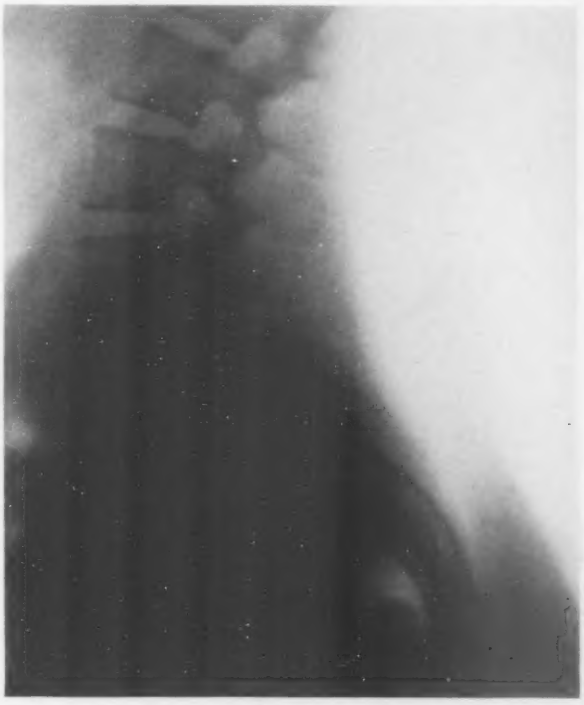

FIG. 2.—Excessive lumbar lordosis. Note V-shaped widening of intervertebral spaces. Intra-abdominal vertebral and sacro-iliac tenderness. Incapacitated as school teacher for seven years by abdominal pain and tenderness. Had futile appendectomy and cholecystectomy. Relieved by corset and novocaine injection of intercostal nerves.

The condition is quite analogous to the strain and tenderness which accompany the breaking down of the arches of the feet. It occasionally happens, that tenderness and evidence of ligamentous strains may be encountered in feet that have unusually high arches at the beginning of their breaking down process. Similarly I believe that exceptionally a fairly normal lumbar curve which is tending toward increased lordosis will exhibit the tenderness of undue ligamentous strain, even before a physical examination or a lateral skiagram of the spine, will demonstrate any undue lumbar lordosis. On the other hand, completely flat feet and excessively lordotic spines may be free from tenderness. It is therefore evident that the degree

of lumbar lordosis is not necessarily the true index as to the amount of tenderness.

Midline tenderness is most easily demonstrable a couple of inches above the umbilicus where the convexity of the lumbar lordosis most nearly approaches the anterior parietal wall. Slightly above this point it is not possible to depress the muscles in the intercostal angle sufficiently to palpate the more deeply situated vertebræ. The tenderness extends down to, and includes, the sacrolumbar joint, but does not extend more deeply over the brim of the pelvis.

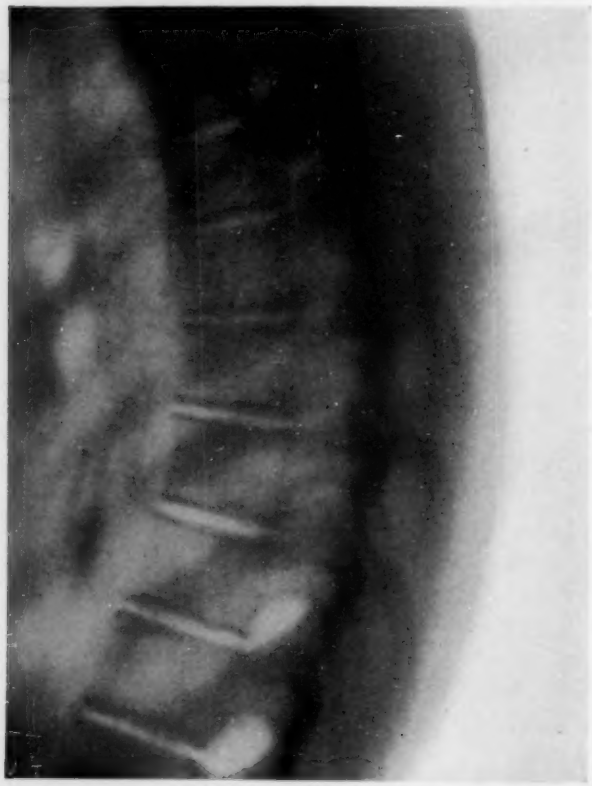

FIG. 3.—Round shoulders compensatory to the lumbar lordosis of Fig. 2.

It is interesting to note how little deviation from the routine method of palpating the appendix is necessary to evoke spinal tenderness. On making deep rotary palpation over McBurney's point with flat fingers, the examiner needs to exert only a little push toward the midline in order to elicit tenderness; which, if he is not aware of having touched the sensitive lumbar vertebræ with the tips of his fingers, he is very apt to mistake for tenderness under the pulps of his fingers and therefore erroneously regard it as appendix tenderness.

Although patients describe their spinal tenderness in diverse terms, yet the tenderness appears to be of a special characteristic type, because I have frequently made contrasts between it and other co-existent and diverse forms of visceral or parietal tenderness, and intelligent patients have always been able to distinguish the spinal tenderness from other types of tenderness. I emphasize the characteristic nature of this tenderness because it is of exactly the same nature as tenderness which can be elicited over an isolated linear area in each iliac fossa, in almost every case of vertebral tenderness. The situation of these linear areas of tenderness correspond exactly to the position of the superior sacro-iliac joint line. In discussing the anatomy of the sacro-

iliac joint with clinicians, I find that all of them are familiar with the fact that this joint presents one aspect posteriorly in the buttock region and a second aspect anteriorly within the true pelvis, but that very few of them are aware of the third or superior aspect located in the iliac fossa. (Fig. 9.) The anterior ligaments, joining together this third or superior aspect of the sacro-iliac joint, are subjected to undue strain in cases of excessive lumbar lordosis, and they exhibit tenderness of the same character as anterior vertebral ligaments.

Tenderness of the superior sacro-iliac joint is located in a line parallel with and about one inch away from the lumbar vertebræ and extends from about the level of the anterior iliac spine upward for a distance of two inches. In patients having a very thin relaxed abdominal wall, as in feeble multipara, it is possible to palpate the depths of each iliac fossa both to the outer side of the superior sacro-iliac joint line, and between the joint line and the vertebræ without finding undue tenderness even when the joint line and the lumbar vertebræ are very sensitive. In patients with thick or tense abdominal walls, it is not possible to demonstrate the absence of hyperæsthesia between the sacro-iliac joint and the lumbar vertebræ.

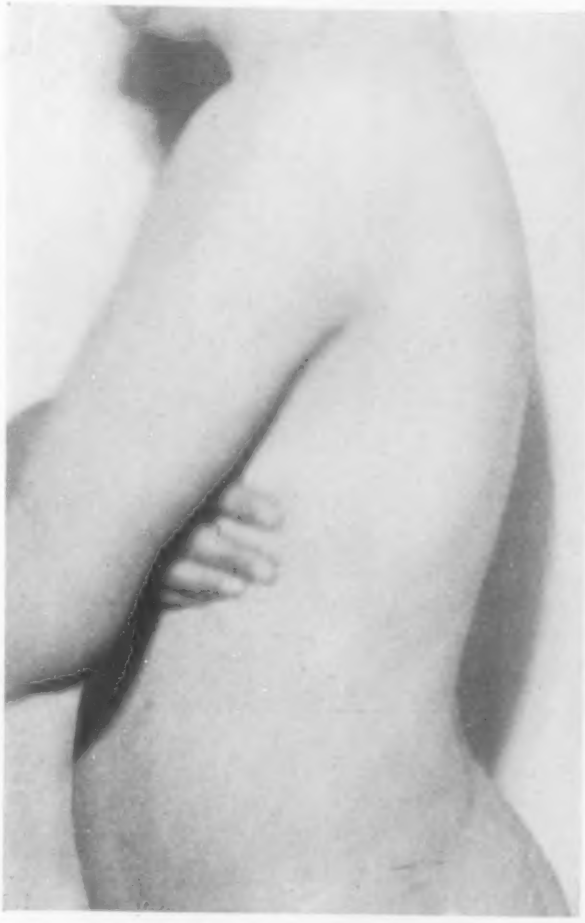

FIG. 4.—Hollow back and round shoulders. Same as Figs. 2 and 3.

A pin passed at right angles through the anterior abdominal wall of the cadaver at McBurney's point will strike the superior sacro-iliac joint line or within one-quarter inch of it. It is therefore obvious that the linear tenderness of a sensitive right sacro-iliac joint in part lies directly beneath McBurney's point and in part corresponds very closely to the tender point which Morris describes as finding by pressing deeply upon the abdomen about "an inch and a half to the right (or left) of the navel and a trifle

caudad" and which he believes is due to hypersensitiveness of the fused second and third lumbar ganglia. He believes that tenderness of this sympathetic ganglion when it is found on the right side only is the most valuable single sign of the presence of a chronic appendicitis, but when the tenderness is bilateral it points to an irritative or suppurative lesion in the pelvis. It is also interesting to note that Hunner finds deep tenderness "at a point about one inch to one side of and one inch below the navel," which he believes is located in the ureter where it crosses the pelvic brim and is indicative of a ureteral stricture. He states that "this sign has led to countless futile appendicitis operations even when it was present over the left ureter alone." He usually finds that both ureters are tender. In passing, it is interesting to note that some of the "vague or complicated abdominal or pelvic pains" with widespread referred pains which Hunter describes in his various papers as occurring in ureteral stricture, are very similar to the pains which I find in some cases of "intercostal neuralgia." I believe the tender areas variously described by Morris, Hunner and myself are probably all one and the same thing. It is also a not uncommon event for the general practitioner who discovers this deep-seated area to ascribe it to tenderness located in the appendix itself and because of the tenderness being rather acute, he sends the patient to the surgeon for a hurry-up appendectomy even though the temperature, pulse and leucocytes be normal with absence of spontaneous pain and rigidity. Fortunately this hypersensitive area is nearly always bilateral and the importance of its presence at McBurney's point on the right side is completely annulled by exactly similar findings on the left side.

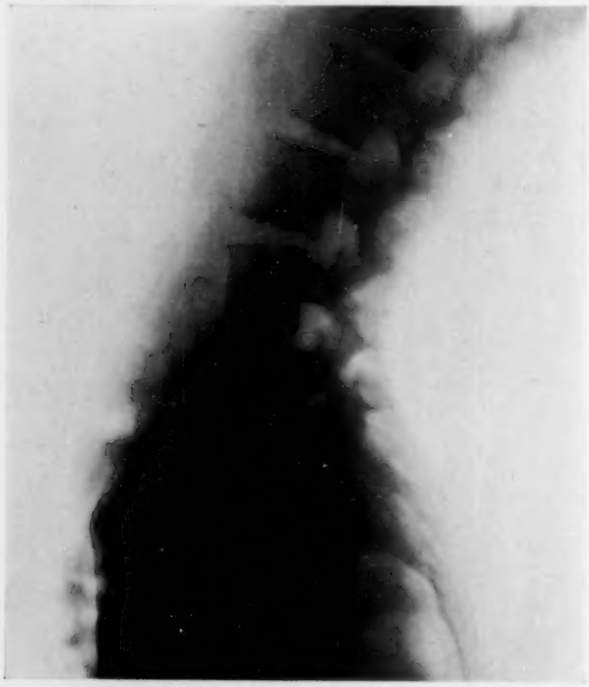

FIG. 5.—Lumbar lordosis with V-shaped intervertebral spaces. Sensitive lumbar vertebræ and sacro-iliac joints. Anterior parietal pain and tenderness began during puerperium and persisted every day for two years when a simple supporting corset was applied. Symptoms disappeared in two weeks and had not returned after seven months.

Furthermore the type or quality of the tenderness over these areas corresponds very closely with the commonly associated tenderness of the vertebral bodies. In my experience this area of tenderness has assumed importance only because of the need to differentiate it from chronic or subacute appendi-

citis. In itself it is of absolutely no value as a sign for or against the presence of appendicitis. It is frequently found in patients from whom the appendix has been removed. Usually the patient does not have any subjective symptoms associated with this tender area. He, or rather she, seeks medical advice because of some other condition and the linear tenderness is first discovered during the systematic palpation of the abdomen.

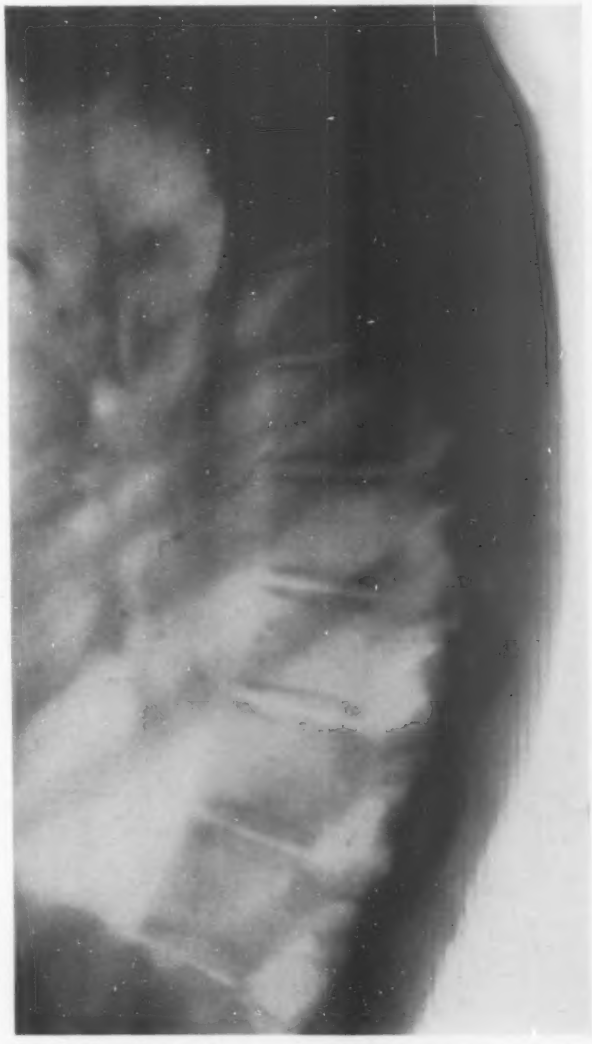

FIG. 6.—Round shoulders compensating for exaggerated lumbar lordosis shown in Fig. 5.

Whether or not I am correct in assuming that chronic strain is responsible for the one elongated midline area and the two shorter lateral areas of tenderness, the clinical fact remains that tenderness of these three deep-seated areas habitually occurs independently of any intraperitoneal lesion. Tenderness in any one of the three areas is almost invariably associated with tenderness in the other two areas.

Any clinician who is not familiar with midline tenderness will find, on reviewing his method of examining abdomens, that he habitually fails to palpate over the midline. I have been very much interested in observing the routine followed by various consultants in palpating an abdomen by going down one side and up the other without touching the midline. They seem to have in mind and conscientiously attempt to palpate liver and gallbladder, duodenum, right kidney, appendix, right ovary and then left ovary, sigmoid, left kidney, and spleen, but they ignore the midabdomen.

Dr. B. P. Widemann has been of great assistance in studying lumbar lordosis from a röntgenologic point of view. He uses the same technic in every case. The patient stands with one side against an erect Potter-Bucky

diaphragm at a focal distance of thirty inches. Cotton pads are carefully adjusted to fill in the space between the Potter-Bucky diaphragm and the lateral wall of the abdomen. Immobilization of the body to the diaphragm is obtained by encircling the body with four-inch wide linen straps at the levels of the hip-joint and of the middle of the chest. Care is exercised to prevent distortion.

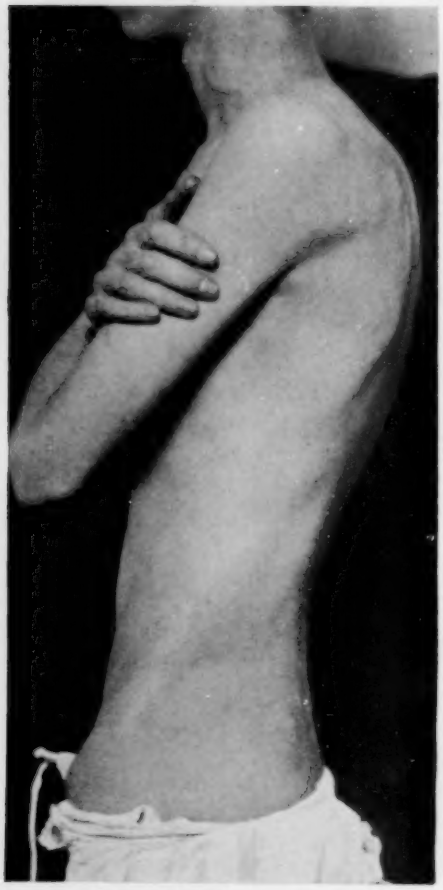

FIG. 7.—The round shoulders and hollow back of same case as Figs. 5 and 6.

The sacro-iliac joint strain is due to sacral participation in the lumbar lordosis. The lumbo-sacral joint line is widened in front and the entire bony pelvis is rotated more or less on a transverse axis, so that the pubes is tilted downward and the sacrum backward and upward. (Fig. 2.) These changes result in an abnormal line of weight-bearing at the sacro-iliac joints.

The mechanism of weight-bearing by the lumbar vertebræ themselves is also disturbed. Normally each vertebræ bears its superimposed weight on three points of support, *viz.*, the body and the two articular processes. In exaggerated lumbar lordosis that portion of the weight normally borne by the front half of the vertebral body is transferred from it to the posterior half of the same body and its two associated articular processes.

Excessive lordosis in the lumbar region is commonly associated with a variable degree of compensatory kyphosis (round shoulders) in the thoracic regions. (Figs. 3, 4, 6 and 7.)

These various changes in the antero-posterior curvature of the spine may be present without any subjective symptoms and without trans-abdominal tenderness of the lumbar vertebræ. Usually, however, the bodies of the lumbar vertebræ are tender and the majority of the patients have backache.

In many of the patients with exaggerated lumbar lordosis the dominant symptoms are those of intercostal nerve irritation, more or less widespread, but manifested mainly by pain and tenderness over the abdomen particularly in the right lower quadrant.

In a recent paper † I pointed out that intercostal neuralgia of the anterior

† Carnett, John Benton: Surgery, Gynæcology and Obstetrics, May, 1926, pp. 625–632.

abdominal wall is a very common affection that frequently leads to erroneous diagnoses and to futile intra-abdominal, pelvic and genito-urinary operations. I will not repeat here an original test and various clinical examinations which I described in that paper to differentiate intra-abdominal lesions from neuralgic pain and tenderness of the anterior abdominal wall.

I am convinced that exaggerated lumbar lordosis is a common cause of neuralgia of intercostal and first lumbar nerves, but I have been unable to demonstrate the mechanism by which it is produced. It is possible that increased weight-bearing at a disadvantageous angle leads to irritation of the joints formed by the articular processes and probably also of the posterior part of the joints between adjacent vertebral bodies. Any periarticular exudate resulting from irritation or chronic inflammation of these joints would be deposited in the immediate vicinity of the spinal nerves as they make their exit from the intervertebral foramina. This explanation may prove true in some cases but in many others the abrupt changes in severity of symptoms, the relief afforded by rest, and the absence of rigidity of paraspinal muscles indicate some other mechanism.

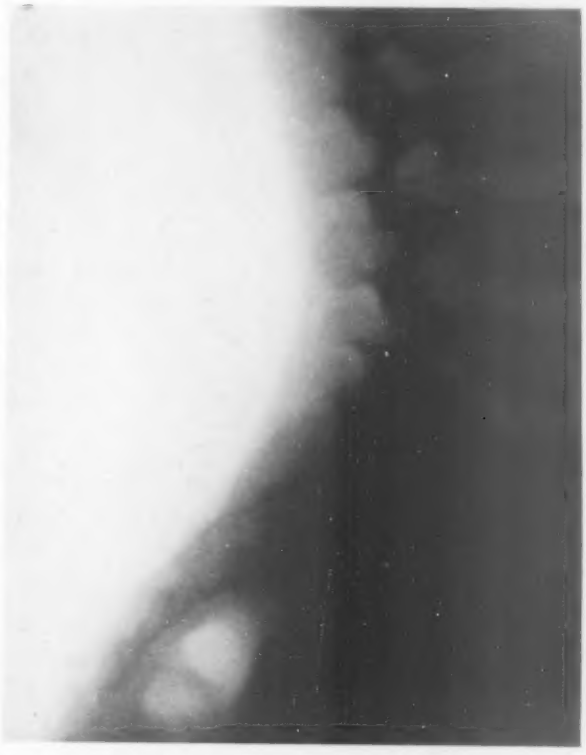

FIG. 8.—School boy, age twelve years. Intra-abdominal vertebral and sacro-iliac tenderness. Repeated attacks of anterior parietal pain and tenderness erroneously diagnosed "recurrent appendicitis."

In making its exit from an intervertebral foramen each intercostal and lumbar nerve passes at a right angle across the edge of a ligamentum subflavum. It is quite conceivable that the instability of a lordotic spine results in excessive intervertebral movements with undue friction between each nerve and its adjacent ligament.

Although numerous nerves may be involved, the twelfth intercostal and first lumbar are usually the ones most affected, causing pain and tenderness in the lower abdomen suggestive of appendicitis or tubal disease.

In the treatment of my cases of chronic strain of the lumbar vertebræ I have had hearty coöperation from my orthopædic friend, Dr. DeForrest

Willard. The milder cases of lumbar lordosis may go through life untreated without symptoms.

The ideal treatment is prevention or early correction of beginning excess lordosis. I have had several children referred with a diagnosis of chronic or recurrent appendicitis in whom all the symptoms were due to excessive lumbar lordosis (Fig. 8) and intercostal neuralgia. Several of this group have recently begun taking corrective muscular exercises under Doctor Willard's supervision. It is too early to note improvement in them but Doctor Willard states that he has had excellent results in similar cases. Correction of the hollow back in children may favorably influence, but is not likely to overcome the coexistent visceroptosis.

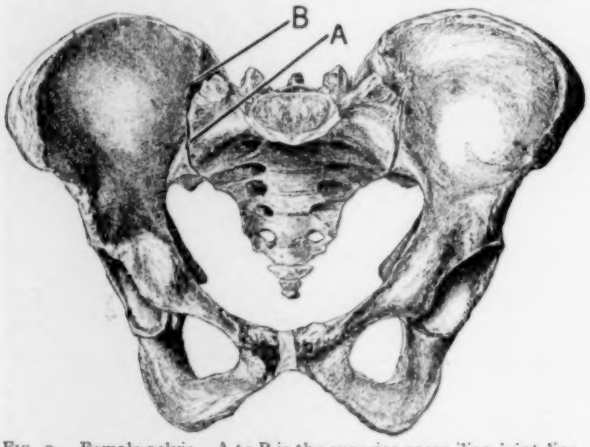

FIG. 9.—Female pelvis. A to B is the superior sacro-iliac joint line, located in the iliac fossa. (Modified from Piersol.)

Patients who first appear during a hyperacute stage of intercostal neuralgia may have to be treated by rest in bed, by removal of toxic foci and by sedatives pending partial subsidence of pain and tenderness. Patients in the subacute stage require a spinal support to relieve the strain on the lumbar vertebræ. This purpose is best accomplished by a corset with firm stays extending from the sacrum upward over the short ribs posteriorly and with a pad over the lower abdomen. I believe that the relief of subjective symptoms obtained by the use of pads and belts in visceroptosis is usually due to relieving strain on the lumbar spine rather than to any elevation of viscera. Corset pressure must be gentle at first, otherwise it will not be tolerated, because of the neuralgic tenderness of the anterior abdominal wall. Relief of subjective symptoms of backache and neuralgia may be attained without any noteworthy change in the lumbar curve.

Even when the upper intercostal nerves are irritated long corsets are not indicated. Correction of the lumbar strain automatically relieves the stress in the dorsal region. Subjective symptoms are relieved promptly and completely in some cases, but are very refractory in many others. In obstinate cases baking and massage of the back are helpful. The milder chronic cases and the acute cases after subsidence of acute symptoms should take systematic exercises directed primarily to strengthening the abdominal muscles and they should endeavor to hold their spines in the corrected position.

FRACTURES OF THE TRANSVERSE PROCESSES OF THE LUMBAR VERTEBRÆ*

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There is apparently considerable difference of opinion in regard to the frequency, importance and method of treatment of fractures of the transverse processes of the lumbar vertebræ. From the scarcity of references in the literature one would think the lesion either rare or of little moment. Some even suggest that it is non-existent, the X-ray appearance being due to bony abnormality. Lumbar pain is often obscure and this may be one factor in it. The recognition of this lesion may rule out more serious conditions, *e.g.*, tuberculosis of the spine, chronic arthritis, and affections of the lumbo-sacral region. A knowledge of the amount of disability caused is important for one thing in estimating insurance loss, as many of these are compensation cases. Some state that prolonged immobilization or more radical procedure is necessary, apparently feeling that they are dealing with an injury followed by considerable disability. In this paper I wish briefly to review the literature and our experience in ten cases at the Surgical Clinic of the Beekman Street Hospital.

Frequency.—Ehrlich,[1] in March, 1908, reported what he considered the first case of isolated fracture of a transverse process of a lumbar vertebra to be discovered by X-ray.

Later in the same year, Haglund[2] reported seven cases of isolated fractures of the lumbar transverse processes occurring in his own practice during the previous six years, all verified by radiographic examination. He refers to six similar cases, not, however, verified by X-ray.

Lange[3] added two cases in 1908, one from strain and one from direct violence, and stated that in each case the injury might well have been passed over as a simple strained back. He believed that the pain might persist for a considerable period because of non-union or delayed union, or that the patient through postural changes might acquire persistent scoliosis.

I could find no further reference to this condition in the literature until 1916, when Cotton[4] reported twenty-two cases so listed in their X-ray department. In ten of these he could see nothing which he personally would call a fracture. He believed that one must consider separate ossification centres and old ununited fractures. Six of the remaining twelve cases, he believed to represent irregular ossification or lumbar ribs. Three others he felt might be due to old injuries. In the remaining three, which were recent injuries by direct violence, he concurred in the diagnosis of fracture.

* Read before the Section of Surgery of the New York Academy of Medicine, January 7, 1927.

Bierman [5] emphasizes the fact that the transverse processes of the lumbar vertebræ, especially the first, frequently show anomalous development. He found this in 5 to 10 per cent. of a series of gastro-intestinal patients examined by X-ray.

In a discussion of 134 back injuries in industrial accidents, Sever [7] mentions only one with fracture of the transverse process. This occurred as a result of a direct blow from a falling object, causing fracture of four processes in the lumbar region.

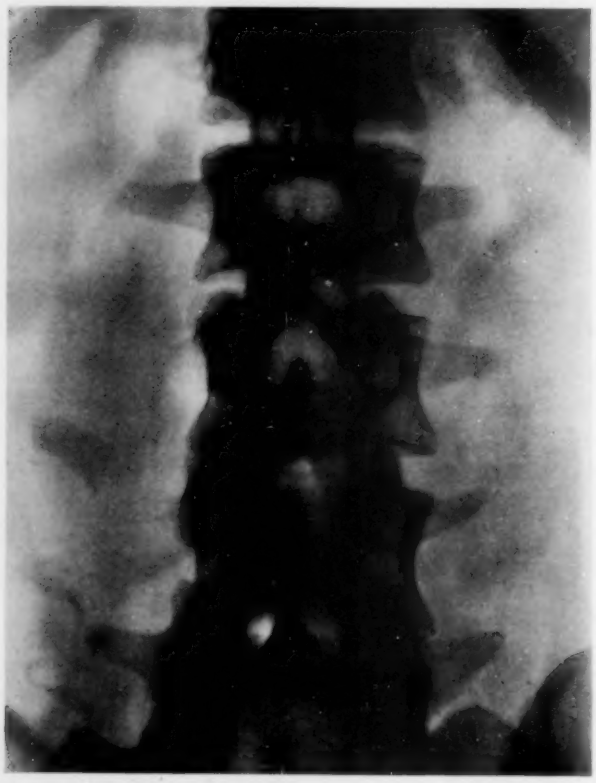

FIG. 1.—Case I. Fracture of the transverse processes of the 3rd, 4th, and 5th lumbar vertebræ, left side.

Stimson [8] states that "fracture of the transverse or articular processes occurs in combination with other fractures (of the vertebræ) in about one-sixth of all cases, but is rare except in such combination. In the few instances in which it has occurred alone, it was the result of a gunshot injury."

The lesion is not mentioned in such volumes as Scudder's *Treatment of Fractures* or Jones' *Orthopædic Surgery of Injuries*. Among 2200 admissions to Beekman Street Hospital in a period of one and one-half years, it occurred ten times, an incidence of one-half of one per cent. These were all definite fractures. In three of them there were associated fractures of the bodies of the vertebræ. The series included nine males and one female. The ages ranged from nineteen to fifty-five, and four of the patients were twenty-five or under, before which age there may be incomplete fusion of the ossification centres.

Etiology.—This lesion may occur from direct violence or muscular strain. Haglund reported three cases by direct violence and three from muscular strain. Two of the latter occurred while riding bicycles and one while lifting a weight. Of Lange's cases, one was by direct and the other by indirect violence. In the latter case, while lifting, the patient stated that he felt

"something give way in the small of his back." Cotton's three cases were by direct violence.

In the present series all were apparently from direct violence, although in some it would be difficult to prove that muscular strain had not played an important part. Two patients were struck across the back by a falling beam, five had falls from a height striking on the back, and three were struck or run over by vehicles.

Anatomy.—The lumbar transverse processes, according to Cunningham,[9] may be regarded as a series of levers to which muscles are attached. Anteriorly, lie the psoas major and the quadratus lumborum, posteriorly, the sacro-spinalis (erector spinæ O.T.). Between these are layers of the lumbar fascia, which blend at the lateral borders of the muscles and give partial origin to the obliquus internus and transversus abdominis muscles. Between the transverse processes are the vertical fibres of the ligamenta intertransversaria. "At puberty a single epiphyseal centre on each side appears at the extremity of the transverse process. Fusion of these centres is not complete until the twenty-fifth year."

Pathology.—The amount of displacement of the fragments is variable. In my opinion, the fractures are accompanied by tearing of muscles and ligaments with hemorrhage.

Symptoms and Physical Signs.—Severe pain and tenderness were present in the lumbar region in all these patients, but not so localized as to indicate the process fractured. Most of these patients were perfectly comfortable while lying flat in bed, but suffered pain on attempting to turn over or to sit up. In no patient was there any suggestion of root pain as if a nerve were involved. Aside from the patients, in whom there were associated spinal cord injuries, at the time of admission to the hospital, all but one could stand. Resistance of the lumbar musculature, particularly of the sacro-spinalis muscle, was present in all patients. In two, neither of whom presented other evidence of intraperitoneal injury, there was abdominal rigidity for forty-eight hours. This may have been due to injury to the attachments of the lumbar fascia, from which the internal oblique and transversalis muscles arise, or to a hæmatoma in close proximity to the peritoneum. Swelling of the soft parts was mentioned in seven patients, usually marked, and in several was limited to the side on which the radiograph showed the fracture. Ecchymosis of the lumbar region was observed in one patient at the time of admission and two others showed a distinct hæmatoma, one of which was later aspirated. In all cases, the normal back movements were restricted because of pain. There was no deformity except from the swelling of soft parts. Five patients showed contusions or abrasions of other parts of the body and one had a fractured rib.

Diagnosis.—The diagnosis can be definitely made only by radiograph. In these ten patients, there were thirty-two transverse processes fractured, seventeen on the right side and fifteen on the left side. Two patients had a fracture of one process only, four had three processes fractured, two had

four and two had five. In eight patients all the fractures were on the same side of the spine, while in the other two, there were three fractures on one side and two on the opposite. In both these latter patients there was also a fracture of the body of a vertebra. In three patients the left side alone was involved and in five the right. The first, second, and third lumbar vertebræ each showed fracture of the transverse process eight times, the fourth six times and the fifth twice.

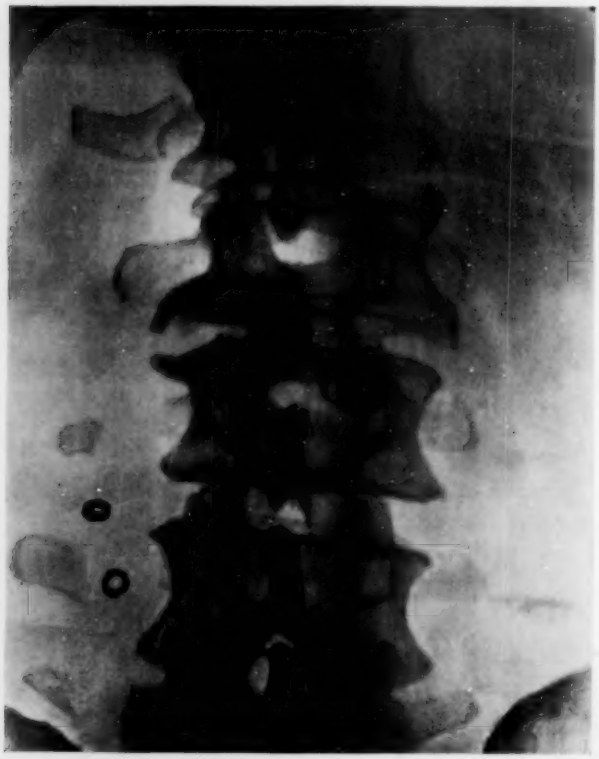

FIG. 2.—Case VII. Fracture of the transverse processes of the 3rd, 4th, and 5th lumbar vertebræ, left side; hypertrophic arthritis in lumbar region.

Prognosis.—The length of disability in these cases is particularly important because the majority of them occur in the class of people covered by compensation insurance. Cotton thinks that this fracture is often overvalued in legal settlement. He states that, "no case has yet come to my attention with apparent fracture of the transverse processes (who did not have a claim on anyone) in whom this fracture seemed to cause disability, beyond that ordinarily associated with contusion or sprain of the back." "There does not seem to be any data on which to base any idea of any permanent disability from this cause."

Sever, in discussing injuries to the back in industrial accidents, states that, "there is a peculiar mental state, analogous to that so often seen in people suffering from litigation neurosis, to be observed in many of these cases, which in my opinion delays their recovery." He found the average disability in thirty-seven cases of contusion of the back to be 6.3 months.

I feel strongly that the duration of disability in these cases may be materially reduced if the patient is not allowed to appreciate that any fracture is present. We are careful not to discuss these cases before the patient and to pronounce them bruises or sprains in talking of his injury with him. In fact, I believe it is the tearing of muscle and ligament with extravasation of blood and the resulting scar tissue, which causes the symptoms and disability, rather than the existence of the fracture.

In forecasting the future, one must also take into account previous conditions of the spine, such as structural anomalies or pathologic changes which make the spine less resistant to injury or slower in repair. The patient may have thought himself well previous to the injury, and yet suffer prolonged disability from insult to a previously existing inflammatory process, *e.g.*, chronic hypertrophic arthritis. While the radiograph may show recent fracture of transverse processes, the patient may make no complaint of this injury. We have had such a patient in our wards since this series was compiled. Following a fall from a height a man fractured both os calces and the transverse processes of the first and second lumbar vertebræ. After the usual rest in bed, on sitting up he complained of pain in the back, out of all proportion to what we have usually seen in these cases. Examination showed all his pain and tenderness to be in the lower, rather than the upper, lumbar region. In going over the radiographs, we found an extensive hypertrophic arthritis with spur formation in the lower lumbar region. He claimed that, although he performed heavy labor, he had never had any discomfort in the back until the present injury. I believe his condition was due to an acute exacerbation of the arthritis following injury, rather than to the fractures. When seen five months after his injury, he had no symptoms nor limitation of motion referable to the back.

This brings up an important point from the industrial and economic view. Many corporations now require a physical examination before employment. The discovery of the presence of a hernia in this examination is said to have been a great saving in after claims. How much more important would be a careful back examination of those who are to be engaged in heavy labor, together with a history of any previous complaints referable to this region. Back injuries are common, malingering is probably quite rare, but many men undoubtedly get credit for it when a mild accident upsets a compensated structural difficulty or activates quiescent disease or injury and thereby initiates disability. Routine radiographs of the spine before employing men over forty at hard labor is more than we can expect to obtain, but I believe would be a distinct economic saving to the employer.

Except for the two cases with accompanying spinal cord injury, the hospital stay for these patients ranged from two to thirty-two days, with an average duration of sixteen days. That is, they were able to walk about in this time, though not to return to their usual work.

In regard to the healing of the fracture, Cotton states that it "may not improbably unite by fibrous union without persistent symptoms."

Osgood[6] states that "fractures of the transverse processes recover if immobilized, but often remain ununited and painful if not recognized and not treated.

In Case VIII, a radiograph taken twelve weeks after injury showed distinct bony union of the third transverse process and suggestive bony union in the first and second; while one taken six months after injury showed

apparently solid union of all fractures. Another case, not considered in this series, shows bony union eight months after injury.

Treatment.—The treatment advised has been varied. Lange states that in his cases, rest in bed, strapping, salicylates, and potassium iodide failed to help, but that the galvanic current gave some temporary relief. Cotton's three cases were all put up in plaster jackets, but the duration of this immobilization is not stated. Osgood suggests complete immobilization for six weeks. Physiotherapy in the form of heat and massage is our routine treatment, commencing usually as soon as the diagnosis is made. The static machine is substituted for manual massage when the patient is allowed out of bed. All patients were put to bed immediately on admission and in most of them the pain was severe enough to require one dose of morphine. Three patients were strapped with adhesive plaster immediately after being radiographed. A fourth, after being in bed for three weeks, was strapped before getting up and the strapping left on for two weeks. Both longitudinal and transverse strapping were employed. In other words, these patients were treated in the same way that we would treat a simple contusion or sprain of the back. With a patient in bed, I believe pain will act more efficiently to immobilize the ordinary injured back than any artificial means we ordinarily use.

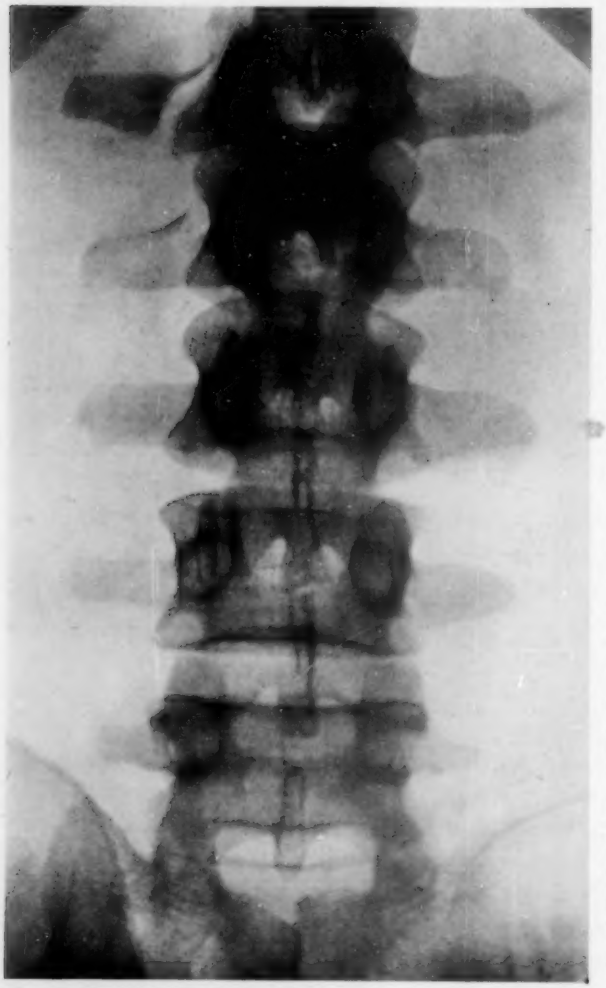

FIG. 3.—Case X. Fracture of the transverse processes of the 1st, 2nd, and 4th lumbar vertebræ, right side.

CASE REPORTS

CASE I.—A. F., male, age sixteen, occupation, clerk. Admitted to hospital, December 14, 1924, having been knocked down by an automobile. Physical examination showed tenderness over the lumbar region, most marked at the level of the second lumbar vertebra.

There was slight swelling over the same region. "Unable to stand." X-ray: Fracture of transverse processes, lumbar 3, 4, 5, left. Treatment: Back strapped. Fifteen days later he was discharged to the out-patient department with little complaint of back. He failed to return and could not be traced.

CASE II.—H. A., male, age twenty-five, occupation, elevator helper. Admitted to hospital, August 14, 1925, having fallen six stories in an elevator shaft striking on back. Physical examination showed contusion over lower lumber region and entire loss of motive power and sensation of both legs. X-ray: Fracture of transverse processes, lumbar 1, 2, 3, 4, left. Backward dislocation of twelfth dorsal vertebra. Operation: Laminectomy. Died of lobar pneumonia thirty-three days after injury.

CASE III.—H. R., male, age forty, occupation, iceman. Admitted to hospital, September 16, 1925, having fallen to the ground from his wagon, striking on his back. Physical examination showed pain in the right side and restricted breathing. X-ray: Fracture of tenth rib right in posterior axillary line and of tip of transverse process of lumbar 1, right. Treatment: Chest strapped. The patient showed few symptoms referable to the back and was discharged to the out-patient department after two days, but failed to return.

CASE IV.—H. M., female, age thirty-two, occupation, elevator operator. Admitted to hospital, August 11, 1925, having fallen one flight in an elevator shaft striking on her back. Pain in lumbar region was severe. Physical examination showed marked tenderness over the lumbar region with swelling and ecchymosis on the right side. X-ray: Fracture of transverse process of lumbar 1, right, with arthritis of the spine and sacro-iliac joints. After two and one-half weeks, patient had slight discomfort in right hip region on weight bearing. There was no tenderness in the lumbar region at the site of the fracture. After two months the patient was having only occasional mild discomfort.

CASE V.—E. G., male, age fifty-five, occupation, laborer. Admitted to hospital, October 27, 1925 shortly after a fall of fourteen feet striking his back on a beam. Physical examination showed both legs flaccid with loss of sensation. X-ray: Fracture of transverse processes of lumbar 1 and 2, right, and lumbar 1, 2 and 3, left; fracture of body of first lumbar with dislocation; fracture of articular process of twelfth dorsal and fourth lumbar. Treated on Bradford frame. After one year the paralyzed condition was practically the same.

CASE VI.—S. R., male, age forty-six, occupation, truckman. Admitted to hospital, December 3, 1925, having been caught between two trucks. Physical examination showed considerable ecchymosis over both lumbar regions, more marked on the right. X-ray: Fracture of transverse processes of lumbar 2 and 3 left and lumbar 2, 3 and 4 right. No displacement of fragments. Compression fracture of body of fourth lumbar vertebra. Treatment: hæmatoma aspirated. Discharged against advice after two weeks. Advised to remain in bed at home for ten days more and then use a supporting corset. Two months after injury he was coming to hospital regularly for physiotherapy and improving steadily.

CASE VII.—J. W., male, age fifty-four, occupation, longshoreman. Admitted to hospital, January 7, 1926, having had a beam fall partially across his back. Patient complained of extreme pain in back immediately. Physical examination showed tenderness and swelling over whole lumbar region. X-ray: Fracture of transverse processes, lumbar 3, 4 and 5, left; marked hypertrophic arthritis in lumbar region; lack of union of transverse process of lumbar 1. Course: After three weeks patient had no complaints referable to back and was allowed out of bed, at which time his back was strapped. This was left on for two weeks. He was last seen eight months after the injury when he did not complain of his back. He complained throughout of his right shoulder, although the X-ray and examination were negative.

CASE VIII.—O. S., male, age thirty, occupation, laborer. Admitted to hospital, May 13, 1926, having been struck on the back by a falling beam. Physical examination showed marked tenderness over whole abdomen and lower back with moderate swelling over lumbar region. There was abdominal rigidity for the first forty-eight hours. X-ray:

Fracture of transverse processes of lumbar 1, 2 and 3, right. Course: After three weeks the patient was still complaining of pain and tenderness in the back, but when he did not know he was being watched, was seen to walk and bend without apparent discomfort. He was discharged from the hospital after three weeks with the function of his spine limited to 50 per cent. After three months his disability was estimated as 12½ per cent. Six months after the injury, he was running a machine in a factory, where he could sit all the time. X-ray, at this time, showed union of all his fractures.

Case IX.—M. R., male, age nineteen, occupation, glazier. Admitted to hospital, May 13, 1926, having fallen three stories in elevator shaft striking lower back. The pain in the lumbar region was severe. Physical examination showed a larger hæmatoma over the lumbar region more marked on the right side. Abdominal rigidity was present for forty-eight hours. There was macroscopic blood in the urine on admission and microscopic blood persisted for one week. X-ray: Fracture of transverse processes of lumbar 1, 2, 3 and 4, right. After one week the patient was walking without pain. After one month he had no complaints except some pain radiating down right thigh and leg.

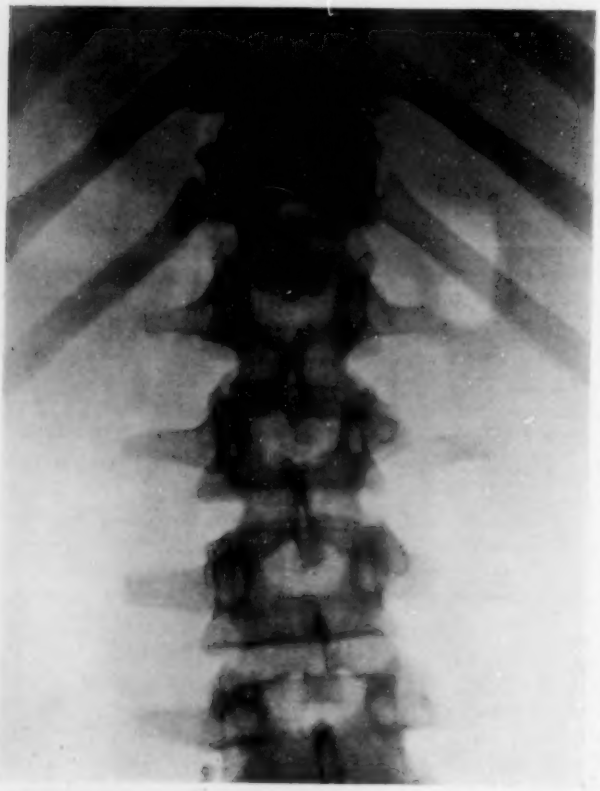

Fig. 4.—E. K., treated since this series was compiled. Fracture of the transverse processes of the 1st, 2nd, 3rd, and 4th lumbar vertebræ, right side. Symptomless and at usual work six weeks after injury.

Case X.—B. R., male, aged twenty-four, occupation, laborer. Admitted to hospital, May 18, 1926, a fire reel having run over his back. Claimed he could not stand and had severe pain on any movement for several days. Physical examination showed a marked hæmatoma with rigidity of the muscles in the lumbar region. X-ray: Fracture of the transverse processes of lumbar 1, 2 and 4, right. Course: Strapped for eight days. Sitting up in bed after one week. Walking in twelve days, but back painful on bending. After six weeks movements in all directions were complete without discomfort. When seen five months after the injury, this was still true. The patient, who had been doing light work from two months on, was then advised that he could take up any occupation he wished.

Results.—Cases II and V are not considered in the results as each had a fracture dislocation of the body of a vertebra. The patient in Case III was discharged to the dispensary two days after injury with no treatment except chest strapping for a fractured rib. It has been impossible to trace him. The patient in Case I was discharged to the dispensary after fifteen days with

a note that there were few symptoms referable to the back. He, likewise, did not return and could not be traced. The patient in Case IV received physiotherapy for two months and after that time, claimed to have only occasional mild discomfort. The patient in Case VI after two months was reported as improving steadily under physiotherapy. The patient in Case VII had no complaints referable to back at the end of three weeks and after a month, records showed normal motion of the trunk on the pelvis, except for some limitation of extension. After seven weeks, however, extension was also said to be complete and without discomfort. He was last seen eight months after the accident, when he was working and did not complain of his back.

The patient in Case VIII was discharged from the ward after four weeks with pain still present and an estimated back function of 50 per cent. After three months his disability was estimated as 12½ per cent. The entire back was held in extreme rigidity, a great deal of which appeared to be voluntary rather than protective. The radiograph at this time showed marked scoliosis toward the affected side, with apparently bony union of one transverse process and suggestive of bony union of the other two. After six months the patient still complained of pain and showed tenderness and spasm of the sacrospinalis muscles, more marked on the side opposite to the fracture. His previous occupation had been that of laborer with the pick and shovel, but he was then working at a machine where he sat all day. The radiograph showed apparent bony union of all three processes. That is, this patient continued to have pain and disability although the fractures were united.

The patient in Case IX, at the end of four weeks, complained only of some pain radiating down the right thigh and leg. The patient in Case X had no complaints after six weeks and movements in all directions seemed to be complete without discomfort. Settlement of his compensation was made two months after injury and he returned to light work immediately. I saw him after five months, when movement was complete in all directions without discomfort and he was advised to do any work he saw fit. That is, in this series but one patient continued to show marked disability after six months and in his case the fractures had apparently united.

SUMMARY

From these ten cases the following conclusions are suggested:

1. Among injuries to the back, fractures of the transverse processes of the lumbar vertebræ are not rare, although it is to be emphasized that bony anomalies in this region are frequent, and may tend to confuse the diagnosis.

2. The most common cause of fracture of the transverse processes is direct violence. The symptoms and physical signs are those of a severe sprain or contusion of the back.

3. Such fractures are usually multiple. When uncomplicated by body fracture, all the fractures are usually on the same side.

4. The disability caused by the injury is due solely to the associated

contusion or sprain of the back, and the presence of the fracture is negligible as far as prolongation of disability is concerned. Owing to the frequency of traumatic neurosis, it is preferable that knowledge of an existing fracture be kept from the patient.

5. Bony union of the fractured transverse processes is definite in some of these cases.

6. More careful examination of backs should be made before employing men over forty at hard labor.

7. The treatment required is rest in bed, heat and massage. Prolonged immobilization is no more necessary than in any contusion or sprain of the back.

8. In this series patients were able to walk after an average period of sixteen days. Disability over six months is out of the ordinary. The majority should be at work within two months with practically no complaints referable to the injury.

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THE USE OF THE RUSSELL APPARATUS IN THE TREATMENT OF FRACTURES OF THE SHAFT OF THE FEMUR

By Thomas J. Ryan, M.D.

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FROM THE SURGICAL SERVICES OF DR. GEORGE P. MULLER, MISERICORDIA AND UNIVERSITY HOSPITALS

In 1924, Doctor Russell, of Melburne, Australia,[1] published a method of treating fractures of the femur which attracted our attention by its apparent simplicity and the good results claimed for it.

For some years we had been following the usual teaching and used the Whitman abduction case for fractures of the neck and base of the neck, extension with ice tongs for the shaft, especially for the supracondyloid fracture and frequent open fixation with plates for shaft fractures. Simple Buck's extension was used in fractures of the neck if the position was good. We were not particularly satisfied with the results of ice tong traction, with suspension, in fractures of the shaft and preferred open reduction with plating, although we realized that in the comminuted supracondyloid fracture ice tongs offered the best means of obtaining traction. We were, therefore, willing to try this method of Russell's, and since December, 1924, we have treated twenty fractures of the femur. When we first began this treatment we applied plaster cases to a number of them after reduction was accomplished. In a few of these cases the reduction was accomplished by operation either because we were unable to replace the fragments or we desired a better reposition. After our open reductions we applied cases. In reviewing our twenty cases we have found details in which the treatment outlined by Doctor Russell was not entirely enforced and on this account we are reporting only eight cases which had the extension applied when the patients were admitted to the hospitals, and not removed until good healing had taken place. In one case which is now being treated we were able to correct the overlapping, but we could not get the fractured ends approximated. We therefore made a small lateral incision over the fracture, and found a large quantity of muscular tissue between the ends of the bone. This tissue was removed, the fragments placed end to end, and the patient again placed in the Russell extension. An X-ray taken one week after operation showed the fragments in perfect alignment. We expect that we will have a rapid formation of callus in this case. The following case reports show that the average hospital days was forty-eight. One case who was confined to the hospital for 300 days had carcinoma of the prostate gland, and a pathological fracture of the femur. Union occurred in his fracture, but he was totally confined to bed on account of his carcinoma, and for this reason his number of hospital days has not been included in the calculation of the average.

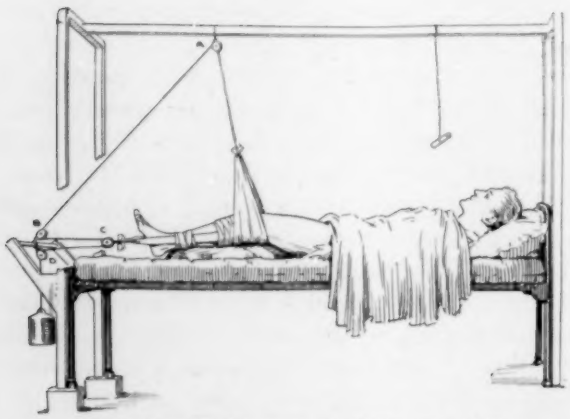
Total disability was determined by their ability to be about without the use of a cane and able to resume their usual occupation with normal function at hip, knee and ankle-joints.

Case No.	Name	Age	Type of fracture	Total disability	Hospital days	End result
5038	N. D.	10	Oblique middle third	9 Weeks	42	No shortening. Normal function.
2282	O. C.	14	Oblique upper third	8 Weeks	39	No shortening. Normal function
2973	T. R.	55	Oblique upper third	Died carcinoma prostate	300	Good reduction and healing without shortening.
549	J. W.	40	Oblique upper third	14 Weeks	57	1.5 cm. shortening. Slight limp. Normal function. in knee.
1717	M. F.	74	Transverse middle third	10 Weeks	55	No shortening. Normal function.
1769	E. P.	54	Oblique middle third	Cannot locate	51	Good reduction and healing on discharge from hospital.
2441	W. R.	13	Transverse middle third	9 Weeks	45	No shortening. Normal function.

The object of the treatment as outlined by Doctor Russell depends upon a natural and comfortable position for the limb with a comparatively small amount of extension to restore the equilibrium in the muscles of the thigh. When the muscles are extended to their normal length and if there is no interposition of tissue, the fragment ends will fall into their natural alignment.

THE APPARATUS

FIG. 1



(British Journal of Surgery, vol. xi, No. 43, 1924.)

(1) An overhead frame to which a pully (A) is attached. This pulley is directly above the extremity and is so placed that a perpendicular from it will fall well down upon the leg—usually between the upper and the middle thirds.

(2) A specially constructed rigging made of wood containing two pulleys (B-D) closely associated.

(3) Buck's extension (which we apply with moleskin) extending to the head of the tibia and fibula with a pulley (c) on the spreader.

(4) A sling of muslin under the knee with twine attached. The twine continues from this sling to the overhead pulley (A) to the first pulley on the rigging (B) to the

pulley on the Buck's extension (C) back to the second pulley (D) on the rigging and the weight applied. The twine is continuous and should be of small diameter with large diameter pulleys to reduce friction.

(5) A soft pillow under the thigh and extending well down under the leg.

(6) Elevate the foot of the bed.

(7) Apply four (4) pounds weight for a child, six (6) pounds up to the age of fourteen (14) and eight (8) pounds for an adult.

(8) Do not apply too much weight.

(9) Have the heel just free of the bed.

(10) Measure the thigh daily.

(11) Examine daily for posterior bowing.

Anatomy of the Thigh [2, 3].—The shaft of the femur is convex in front, except below the neck, where it is slightly concave. On the posterior surface is the linea aspera, a prominent longitudinal ridge along the middle third, which gives strength to the concavity here. Below the middle of the bone the linea aspera divides into the internal and external supracondylar ridges. It is composed of a head, neck, greater and lesser trochanters, shaft and internal and external condyles.

On the front of the thigh the sartorius, gracilis, and rectus femoris, which unite with the vastus internus, vastus externus and vastus medialis to form the quadriceps, arise from the pelvis and insert into the head of the tibia.

On the back of the thigh the semitendinosus and semimembranosus on the inside are seen to arise from the tuberosity of the ischium, and insert into the head of the tibia while the long head of the biceps arises from the ischium and inserts into the head of the fibula.

The above knowledge of the thigh suggests the wisdom of applying Buck's extension below the knee in order to secure the greatest amount of extension on these muscles which, as it must appear, are the greatest factors in the production of the deformities.

The Physiology of Muscles.[4, 5, 6].—A muscle is an organ composed of many thousands of muscle fibres bound together by connective tissue and surrounded by a sheath of the same tissue.

Muscular tissue, when acted upon by a weight, extends quite readily, and when the weight is removed, it regains its original form by virtue of its elasticity. In our bodies the muscles stretched from bone to bone are in a state of elastic tension. If a muscle is severed by an incision across its belly the ends retract. A muscle that is in a state of elastic tension contracts more promptly and more effectively for a given stimulus than one which is entirely relaxed. The extensibility of muscular tissue as compared with dead elastic bodies is quite striking. With regard to the latter, it is well known that the amount of strain that the object undergoes is proportional, within the limits of elasticity, to the stress put upon it; however, the former acts in a very different manner, *i.e.*, the greatest amount of extension occurs with the application of the first weight and decreases proportionally with new increments of weight. Haycraft calls attention to the fact that under normal conditions the physiological extension of the frog's muscles in the body is equal to that

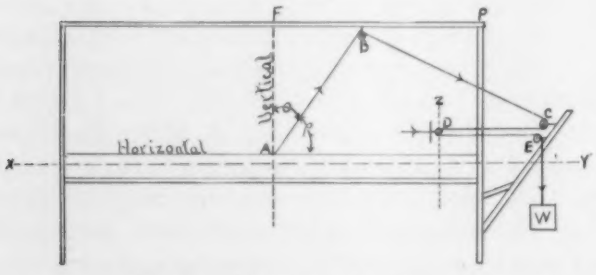
produced by a weight of ten to fifteen grams and that when the excised muscle is extended by weights below this limit, it follows the law of dead elastic bodies, giving equal extensions for equal increments of weight. It has been shown that the extensibility of a muscle is greater in the contracted than in the resting state. Under normal conditions in the body a muscle is made to contract by a stimulus received from the central nervous system through its motor nerve; however, Howell and Haller have shown that skeletal muscle possesses the properties of independent contractility and independent irritability (Bernard), the former being due to active processes developed in its own tissue and the latter the result of artificial stimulation to its own substance. Whether the nerve cell or gland cell may be made to enter into its specific form of activity by the direct application of an artificial stimulus is still undetermined, but the reactions have occurred with the motor nerves severed.

In addition to the properties of contraction and of relaxation the living muscle exhibits the phenomenon of "tone." By muscle tone is meant a state of continuous shortening or contraction which under normal conditions is slight in extent and variable. This condition is supposed to be dependent upon subminimal nerve impulses which are being continually sent into the muscles by external influences. It may be beneficial in maintaining the nutrition of the muscle, regulating the heat of the body and causing a rapid response for a sudden voluntary contraction. The sensory and motor nerves therefore play an equally important rôle in the production of muscle tone. Royle has shown that the sympathetic nerves which supply the voluntary muscles have control over "plastic tone," whereas the medullated nerves control the "contractile tone." Both of these properties being the result of reflected sensory stimuli and in the production of deformity in fractures are due to the effort of the patient to maintain stability of the injured limb and sensory stimuli from the irritation of the fractured ends.

Is it not essential then that the thigh should be immediately returned to a condition of muscular equilibrium by extension and a comfortable position as soon as possible after fracture occurs?

THE MECHANICS OF THE EXTENSION

FIG. 2



F. = force.
W. = weight.
f. = friction of cord throughout system.
f = pulley friction.

$FAB = W - (f. f_B f_C f_D f_E)$

$FAF = W - (f. f_B f_C f_D f_E) \cos \theta$

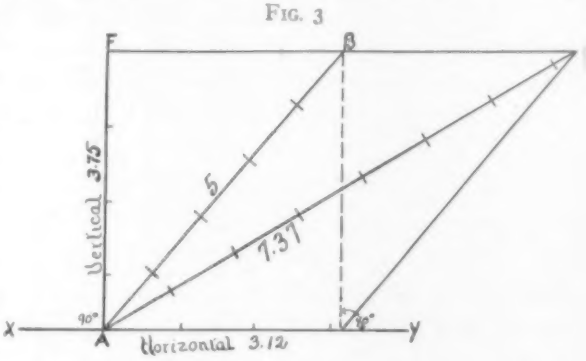
$FAY = W - (f. f_B f_C f_D f_E) \cos \rho$

FAF and FAY may be solved graphically as shown in Fig. 3.

Explanatory.—Make length of line AB in the above figure contain the same units of length as there are pounds of force along AB; the number of units in AF will give the vertical force acting at A, and the number of units of length in AY will give the horizontal force acting at A.

If it is required that FAB shall give a greater pull along AY, move pulley B. nearer to P. If greater vertical pull along AF is required of FAB, move pulley B. nearer F.

FIG. 3



With the horizontal force computed a second parallelogram may be constructed and the resultant (7.37) of these two forces is seen acting in the axis of the femur and is greater than the weight applied.

To get maximum FDC-E, the lines DC and DE must be parallel to XY and when this is so there will be no vertical forces along DZ.

To obtain maximum FAB and maximum FDC-E, the pulleys in the system must be as large as can be conveniently used.

The friction factor, f. may be considered as *practically* zero with small cord, however, f. will increase with the increase in diameter of the cord used. A close-link chain would offer very little f.

The friction factor f_B f_C f_D f_E will be quite small; approximately 5 to 10 per cent. of W.

The Physiology of New Bone Formation.—When a fracture is produced, hemorrhage is the first and most important requisite to healing that occurs. The presence of blood about the fractured ends produces a moderate amount of inflammation and the mixture of blood clot and inflammatory exudate is formed around the fragments. The amount of this exudate determines the amount of future callus.

The blood clot becomes converted into fibrous tissue, and this tissue being acted upon by osteoblasts, which are really fibroblasts with the capacity of producing bone, is transformed into bone.

At the end of two days the intervening substance between fractured ends is granulation tissue. In the course of four or five days this becomes converted into osteoid tissue, which is tissue resembling bone in its structural arrangement with a homogeneous matrix but not possessing any lime salts. The osteoid tissue is scattered in clumps and in the intervening spaces marrow-like tissue develops. Finally lime salts are laid down and the ends are united by fully formed bone.

Part of the new bone may first be formed through cartilage. Areas of cartilage are laid down by the osteogenic tissue of the periosteum and here are invaded by new vessels, converted into osteoid tissue and finally calcified, just as happens in the normal development of bone in cartilage. This method of healing occurs where there is a large gap to be filled and where there is considerable amount of movement.

Healing of the fracture does not occur through the proliferation of the bone cells because these are end products and are incapable of multiplication.

Callus is the mass of new tissue which is formed and it is divisible into three portions, namely, external, internal and intermediate. The external ensheaths the junction, the internal fills the marrow cavity and the intermediate joins the fractured ends.

CONCLUSIONS

1. This method is inexpensive, practical, comfortable to the patient and simplifies nursing care.

2. It is ideal in elderly patients, especially in cases of fractures at the neck when the Whitman case treatment is not desired.

3. Stiffness at the knees occurs less frequently than after other methods; the period of disability is thereby reduced.

4. This simple means of extension is just as efficient in the average case in effecting reduction of the fragments as is tong extension with heavy weights.

5. Occasionally an open operation may be required to disintegrate the fragments from binding muscle.

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HEPATITIS AND ITS RELATION TO CHOLECYSTITIS

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THE MUTTER LECTURE DELIVERED BEFORE THE PHILADELPHIA COLLEGE OF PHYSICIANS, DECEMBER 10, 1926

In several papers written during the last nine years, Evarts Graham,[1, 2, 3] whom I think your Committee honored the year before last as it has me this year, has presented some interesting observations and stimulating suggestions on the pathogenesis of hepatitis and cholecystitis, on the path of infection in diseases of the gall-bladder and on the association of cholecystitis and hepatitis.

In reading these papers and others on the same subject, I am reminded of a saying of the hæmatologist Naegeli, quoted by Aschoff.[4] He writes: "It is a sure sign of the undeveloped state of science if the logical concepts with which it deals are not fixed and when multiple meanings are assigned to the same term." I am reminded of this saying because of the confusion in my own mind regarding the exact meaning of hepatitis and cholecystitis. Perhaps the disagreement and controversies that arise between clinical surgeons, pathologists and physicians, when terms such as hepatitis and cholecystitis are used, are inevitable in the uncertain state of much of our knowledge regarding these subjects and the different conceptions formed when these terms are used.

At the outset it may be well to refer briefly to the observations that have been made regarding hepatitis and cholecystitis by a number of surgeons during the last ten years and to state the conclusions that have been drawn from these observations. Small sections of the liver removed during operations for cholecystitis have regularly shown varying degrees of hepatitis. The lesions are confined largely to the interstitial tissue where the lymphatics are situated. In several instances pieces of liver removed during operations for chronic appendicitis have shown inflammatory changes. Gall-bladders removed for a group of clinical symptoms which are classed as dyspepsia, and in which there was no history of gall-stone colic, have not infrequently shown very slight or no macroscopic lesions, but have shown, under the microscope, slight inflammatory infiltration of the deeper layers of the walls of the gall-bladder. The conclusion has been reached that in many cases of cholecystitis there has been a direct extension to the wall of the gall-bladder from an inflamed liver; that frequently a vicious circle exists between the gall-bladder and the liver whereby each may infect the other; that the infection of the liver from an inflamed gall-bladder is an important factor in the production of cirrhosis; that lesions of the gall-bladder wall that cannot be readily recognized macroscopically give definite clinical symptoms.

I may also add at the outset that anyone having the temerity to discuss hepatitis is thrown on the horns of a dilemma. Either he must discuss the

subject as if hepatitis were a simple entity or he must refer to its very complex and diversified etiology and be carried into a rather loose-jointed and rambling discussion. I must beg your indulgence for the latter method.

I have thought, however, it might be of advantage to bring forward some of the problems that present themselves when the somewhat indefinite word hepatitis is used and to consider the interesting question as to when the reaction of the cells that make up the liver and the gall-bladder reaches the plane of clinical observation. In other words, what degrees of reaction in the hepatic cells or the gall-bladder wall are we justified in considering clinical entities.

There are a few features in the microscopical anatomy and physiology which seem to me pertinent to the discussion. The arrangement and relation of the blood and lymph capillaries to the liver cells are imperfectly understood. There is, however, a fairly general agreement that the portal vein and the hepatic artery spread out into the intricate capillary network which is very intimately related to the liver cells. The Danish zoologist, Krogh,[5] who has made most extensive studies of the anatomy and physiology of the capillaries, considers that the endothelium of the hepatic capillaries is a syncytium with numerous nuclei but without defined cell borders as in the embryonal capillaries, and that the star cells of Von Kupffer, which appear at rather short, regular intervals, are an integral part of the capillary wall. The English histologist, Schafer,[6] believes that what remains of the endothelium of the liver sinuses is represented by these stellate cells.

Blood, entering the liver through the portal vein and hepatic artery, spreads out in a large and complex capillary network, the liver cells being directly bathed by blood and not by lymph, as elsewhere in the body. Besides the polygonal liver cells and the Von Kupffer cells which make up, with the capillaries, the lobules, there is a connective-tissue stroma underneath the serous covering and between the lobules which supports the bile ducts and blood-vessels in their course through the organs. The lymphatic vessels arise from the lymph capillaries in this connective-tissue stroma. Numerous lymph vessels accompany the interlobular branches of the portal vein and numerous lymph vessels accompany the hepatic vein.

From the study of the injection specimens made by Sappey,[7] it seems that a considerable number of fairly large lymph vessels pass along the inferior surface of the liver over the body of the gall-bladder from the adjoining quadrate lobe and the right lobe, to form an extensive anastomosing network of vessels in the neighborhood of the neck of the gall-bladder. Others, derived from the same sources, pass behind the gall-bladder to terminate in the same region. There are apparently numerous anastomosing lymph capillaries between the lymphatics of the liver and the gall-bladder.

The hepatic cells have many and diversified functions. The liver is the great laboratory and storehouse of the body. It is engaged in chemical transformations, demonstrated by the high consumption of oxygen and the production of heat. All three classes of foodstuffs—carbohydrates, fats, and

probably proteins—are built up into large non-diffusible molecules and stored in the liver cells and, by reversible ferments, again changed into soluble forms which pass out into the circulation as needed. The liver cells change the amino acids to simpler compounds, produce bile with its well-known function and play an important part in the coagulation of the blood.

But it is to a function which it shares with the reticulo-endothelial system, in rendering harmless various toxic substances and in the destruction and disposal of living microörganisms and red blood-cells, that I wish to call special attention. The stellate cells of Kupffer, which we have just said are part of the lining endothelium of the liver capillaries, belong to the group of reticulo-endothelial cells which are found in the sinuses and pulp cords of the lymph-nodes, the reticulum and the blood sinuses of the spleen and the capillaries of the bone-marrow. These cells behave in a special way in taking up dyes and other substances and show a pronounced capacity for phagocytosis.

Kupffer showed that the star cells in the liver were loaded with the pigment after the injection of india ink. These experiments have been many times confirmed. Various chemicals injected into the circulation are taken up in the same way by the stellate cells. Certain salts are not arrested; the cells show a power of selection and certain substances are taken up and stored in the liver cells. A number of careful experiments by Roger [8] and others have shown that alkaloids are arrested in the same way in the liver. Some of the alkaloids lose a portion of their toxicity in passing through the liver; some of them are excreted in the bile and, according to Roger and others, the detoxicating effect varies with the abundance of glycogen in the liver.[9] There can be no question of the arrest, storage and destruction in the liver of a number of simple and complex chemical substances.

The action of the liver on microbes is equally interesting. Wyssokowitsch[10] showed, many years ago, that pathogenic and non-pathogenic microbes and the spores of molds were arrested and taken up by the cells of the capillaries and sinuses in the liver, bone-marrow and spleen. The capillaries and sinuses in these regions are great settling basins where all manner of minute foreign substances are deposited and taken up. Although all the cells of the so-called reticulo-endothelial system in these situations are largely concerned in the reactions, it by no means implies that their behavior in the liver, spleen and bone-marrow is the same. The stellate cells of the liver are not the exact equivalent of the endothelial cells of the splenic sinuses, for example. Bacteria are not taken up to the same extent in all the depots of this system of cells. The Kupffer cells of the liver seem to take up and retain large numbers of microbes at an early stage, at least in inoculation with certain microbes and in certain animals.

Bacteria and other foreign substances taken up by the reticulo-endothelial cells in the bone-marrow and spleen are destroyed there or start to grow and invade again the blood stream. But for the material deposited and taken up by the liver cells there is another possibility. The foreign particles may be

excreted in the bile. In Wyssokowitsch's (*l. c.*) experiments he expressly states that the bacteria taken up from the blood stream do not pass out by the excretions and secretions of the body. But it has been proved over and over again, during the last forty years since he made his precise and interesting observations, that bacteria injected into the portal vein or the systemic veins, if injected in sufficient dosage, regularly enter the bile. Nichols [11] recorded in 1916 a number of carefully carried out experiments and reviewed and criticized the work done up to that time by Fütterer, Chiarolanza and Koch. He injected varying doses of typhoid bacilli and cholera vibrios into the systemic veins and into the portal vein in immune animals and in animals in which no immunity had been established. He found fairly large doses were necessary to make bacilli appear in the bile and a larger dosage was required in the systemic veins than in the branches of the portal vein. He records several instances in which the results were strikingly inconstant; the same dosage injected in a portal vein in one instance showing numerous colonies cultured from the bile and in another showing not a single colony. There were marked individual variations in elimination. Immune animals showed a greater power of excreting bacteria in the bile than non-immune animals. Rapid agglutination *in vivo*, deposition in the liver and corresponding elimination are suggested as explanations. The bacilli began to appear in the bile with astonishing rapidity, being found in two to three minutes after portal injection and the first plates showed the largest number of colonies.

The reticulo-endothelial cells are concerned not only in taking up foreign bodies and, through the action of various intracellular ferments, modifying or destroying them, but they are also concerned in furnishing and secreting various substances which aid in the destruction of microörganisms and in the neutralization of their toxins. Metchnikoff [12] wrote that it was probable that the macrophages (and in his macrophage system is found the first suggestion of a reticulo-endothelial system) represented the principal source of antitoxin. Since then it has been shown by Hahn, Von Skramlik and Huenermann [13] that the perfusion of the liver of a sensitized animal absorbs the corresponding antigen. The stellate cells of Kupffer are assumed to be the active agents in the liver. Ehrlich and Morgenroth [14] observed that in dogs poisoned by phosphorus, with a consequent degeneration of the liver, there was a diminished production of alexin or complement. Although later experiments have not by any means settled these questions, there can be no doubt that the reticulo-endothelial system, and consequently the stellate cells of the liver, play an important part in the taking up and destruction of bacteria and in the altered reaction of the body to introduced bacteria.

If next we turn to the circumstances under which bacteria invade the tissues and settle in the liver and consider that, aside from the fact that in very many infections where organisms are introduced through the skin or through the genito-urinary system, the respiratory system, the mouth, the pharynx and œsophagus, and in which bacteria gain entrance into the systemic

circulation and are widely distributed, there is a large group where invasion occurs through the portal system.

Numerous observers have studied the intestinal flora. We all know that multitudes of bacteria, aërobic and anaërobic, inhabit and flourish in the large intestine and the lower portion of the small intestine, becoming less and less under normal conditions as the upper jejunum is reached. The intact epithelial cells, the protective mucus, the continual movement onward of the intestinal contents, all prevent the passage of bacteria through the intestinal wall. Slight alterations, such as congestion or catarrhal inflammation and demonstrable morphological lesions may, of course, alter conditions so that the intestinal wall is penetrated. Fifty years ago Pasteur and his collaborators showed that the anthrax bacillus which is so fatal for mice and guinea pigs, could be swallowed with impunity. To produce anthrax invasion by the path of the intestine it was necessary to feed the animals powdered glass and sand.

I wish, however, to call your attention to a number of experiments which show that bacteria can pass through the normal intestinal wall. There is mention by Metchnikoff [15] of an unpublished paper by Mitchell on experiments carried out in his laboratory, in which animals were killed by the invasion of anthrax bacilli through the intestinal mucosa, to all appearances normal and in which the animals received no nourishment capable of producing lesions of the intestinal wall. The animals were fed anthrax spores mixed with a pap of bread soaked in milk. In 1894, Macallum,[16] in studying the absorption of iron, showed that leucocytes could pass out into the intestinal lumen, take up foreign material and return through the intestinal wall. He recovered the leucocytes containing stained granules in the liver and spleen. The leucocytes found their way into both the systemic and portal circulation. The next year, Desoubry and Porcher [17] working in Nocard's laboratory, established by numerous experiments that during digestion bacteria of all kinds may pass through the normal mucous membrane of the intestines and may be found during several hours in the chyle and blood. These experiments were made at the suggestion of Nocard, who had noticed that serum obtained from blood withdrawn with every possible care from horses shortly after feeding was, every now and again, contaminated.

In studying the pathogenesis of tuberculosis a number of experimenters have confirmed these observations. During digestion many bacteria pass through the intestinal mucosa with the chyle and are found in the mesenteric glands, in the lymph of the thoracic duct, in the blood current and in the liver, spleen and bone-marrow. The younger the animal the more constant the results. The bacteria find their way through the mucous membrane of the intestinal wall without leaving a trace of their passage. Calmette and Van Steenberghe [18] studied the mechanism of passage. They confirm the observations of Macallum. Polymorphonuclear leucocytes in large numbers pass through the mucosa into the lumen of the bowel normally. They form, with the epithelial débris, a not inconsiderable part of the fæces. Attracted by chemotaxis to the surface of the intestine and the secreting surfaces of the

glands, hundreds of thousands pass out daily. Bacteria in the intestinal lumen are taken up by some of these phagocytic cells. During digestion, especially when bacteria are intimately mixed with the food, the bacteria are carried by the leucocytes back through the intestinal wall to enter with the minute globules of fat or the salts of fatty acids, made by breaking up the fats, into the lymph vessels of the villi of the small intestine and hence to the systemic circulation or they find their way to a less extent into the capillaries of the villus and to the portal circulation.

The bacteria taken in may be destroyed by the intracellular ferments of the leucocytes; or the leucocytes containing microbes may be arrested and destroyed in the submucous lymphatics or in the lymph-nodes. However, not infrequently living bacteria reach the liver and are destroyed there. Ford [19] showed that 70 per cent. of the livers removed aseptically a minute or two after death yielded cultures of microbes similar to those found in the intestine. The bacteria seemed to be feeble and attenuated and were slow to grow. The presence of tetanus antitoxin in the serum of individuals who carry tetanus bacilli in the digestive tract has been shown by Ten Broeck and Bauer.[20] They showed spores of tetanus bacilli in 34 per cent. of the stools of 78 individuals in Peking. May it not be assumed that here we have an instance of a reaction being set up by spores absorbed during digestion and carried to the liver? Obviously, the nature of the bacillus, the character of the food, the species of animal, may all cause wide variation. The fatty and waxy coating of the tubercle bacillus, for example, makes it particularly resistant to the intracellular ferments of the leucocytes.

The question as to the reaction set up in the liver by the lodged bacteria is equally interesting. That bacteria, without the signs of tissue reaction, can be found in both the liver and the gall-bladder, has been pointed out by Aschoff.[21] He had a large number of unchosen livers examined for possible inflammatory reactions. He concludes that although staphylococci, streptococci and pneumococci can be more or less frequently demonstrated bacteriologically, yet the gall-bladder and the intrahepatic bile passages may be normal in appearance and therefore that certain bacteria may be excreted through the liver and perhaps through the gall-bladder without a special reaction.

Adami [22] pointed out that, in conditions of congestion and slight chronic inflammation, bacteria may, time after time, pass through the intestinal wall to be deposited and destroyed in the liver. The microbes may not necessarily multiply in the organ. Repeated inroads and repeated destruction of bacteria, however, yield enough toxic substances to produce an appreciable alteration in the parenchymatous cells. He suggested the term subinfection to describe the reaction.

The reaction in the reticulo-endothelial cells, when considerable numbers of microbes are introduced into the blood, has been made the subject of an interesting study by Oerskov.[23] He used more or less virulent strains of streptococcus, staphylococcus, pneumococcus and bacillus coli. He destroyed

the animals at varying intervals from five minutes to several days after the injection. He studied the amount of phagocytosis by the Kupffer cells and the amount of exudation of leucocytes. He found in animals killed after two minutes many cocci in the liver and a few in the spleen. The cocci were largely inside the Kupffer cells. The leucocytes were few. In an hour and a half all the Kupffer cells were packed with cocci and there was a considerable exudation of leucocytes. At the end of six hours the liver contained a great number of cocci and the leucocytes were in great numbers. At the end of eighteen hours there were still a large number of microbes in the liver but many of them stained poorly; the leucocytes were numerous. At the end of eight days the microbes were no longer found. In the destruction of the cocci by the Kupffer cells there was pronounced infiltration of the interstitial tissue with leucocytes; similar results were obtained in studying the destruction of other microbes. None of the polymorphonuclear leucocytes showed evidence of phagocytic activity.

In another series he introduced a fatal dose of pneumococci. The results were similar up to a certain time. The cocci were rapidly arrested and destroyed in the liver by the Kupffer cells, accompanied by the same infiltration of the interstitial tissue by leucocytes, but at the end of twenty-four hours there was a multiplication of the pneumococci. The protective cells were unable to hold in check the microparasites and the body was invaded.

According to the type of organism and the degree of immunity established in the body, not only an exudative inflammation occurs, but various forms of degeneration, necrosis and regeneration, and various forms of productive inflammation, giving the most varied picture of interstitial and parenchymatous lesions.

Chronic productive inflammation of the interstitial tissue is very frequent and leads to a variety of alterations in the liver tissue which are grouped under the general heading cirrhosis.

There is a wide margin of safety in the liver. Rous and MacMaster [24] have shown in the dog and the monkey that three-fourths of the secreting cells of the liver can be shut off without producing symptoms. That considerable cirrhosis can exist without clinical symptoms has long been well recognized.[25]

There can be no question that the liver is concerned in the defense mechanism of the body. It is concerned normally in the disposal of and making innocuous various substances brought to it; in the exaggeration of this function various reactions occur which are shown morphologically. The liver seems to be one of the first organs to react and forms a first line of defense, as it were in the protection of the body against disseminated bacteria.

Many of these reactions pass without evidence of disturbance, such as fever, etc., which can be recognized clinically. There is an interesting hypothesis of Vaughan [26] in this connection. He suggests that when bacteria are destroyed, the general body reaction, the heightened metabolism, the rise of body temperature, all the general disturbances that we group under fever,

are due to the disintegration of foreign bacterial proteins by extracellular ferments. The destruction of bacteria inside the cells may be unaccompanied by these phenomena. The intracellular ferments in the macrophages (Von Kupffer cells) carry perhaps the destruction of the bacterial proteins to a point where the products are much less toxic or build them up into their own protein. The process of intracellular digestion may pass so smoothly that the host may be unaware of the bacterial destruction going on and present no disturbance of the normal working of the body that can be recognized clinically.

The large phagocytic cells of the reticulo-endothelial system have another function. They are concerned in the destruction and disposal of red blood-cells. This has been shown to take place normally in the liver, the spleen, the bone-marrow and the lymphatic nodes. The hæmoglobin is split into an iron-containing portion and an iron-free portion and the liver seems to play an important part in the disposal of both portions. When the red cells are destroyed in excess the iron accumulates in the liver. The iron produced in excess in the reticulo-endothelial system is released into the circulation and stored and slowly secreted by the liver cells. It shares this function of storage with the spleen and takes it over when the spleen is removed.

The disposal of the iron-free portion has been the subject of much study. It is concerned in the production of bilirubin. There has been much controversy over the site of the formation of bile pigment and the production of jaundice. In the well-known experiments of Naunyn and Minkowski [27] the liver was removed from a goose; then the bird was poisoned with arseniureted hydrogen. No icterus followed. In another experiment the liver was not removed. In this instance poisoning by arseniureted hydrogen resulted in an enormous phagocytosis of erythrocytes and the production of jaundice. The text-books of physiology of fifteen years ago record the famous experiments. But as almost all the reticulo-endothelial cells in birds are in the liver, the spleen and bone-marrow containing relatively few cells of this system, these experiments were inconclusive when applied to mammalia.

Later, Whipple and Hooper [28, 29] excluded the portal circulation in dogs, injected hæmoglobin and were then able to show bile pigment in the urine, and Mann and Magath [30] observed the formation of bilirubin in dogs after the removal of the liver. The text-books of physiology of the future will record these experiments. It seems to be established to-day that one of the functions of the reticulo-endothelial system is the production of bilirubin. The Kupffer cells of the liver make up an important part of this system, so that although there is an extrahepatic formation of bilirubin, it by no means implies that the liver is not the seat of a considerable portion of bile pigment production. Of the polyhedral cells of the liver it may perhaps be said that the evidence to-day is against their ever sharing in the formation of bilirubin. They excrete the pigment but do not form it. The bilirubin made by the reticulo-endothelial cells seems to be slightly different from the bilirubin secreted by the liver. Van den Bergh [31] showed that these differences could

be detected by the behavior of the pigment in the diazo reaction. These differences are possibly due to physical changes in the state of the bilirubin in the plasma. The bilirubin acted on by the liver cells diffuses more readily and is more readily oxidized. It gives what is known as the immediate or direct reaction. The bilirubin, as it is formed by the cells of the reticulo-endothelial system, diffuses less readily and is less readily oxidized. It gives a delayed or indirect reaction.

Concerning the secretion of bile salts and cholesterin, little definite is known. It is assumed by Brulé [32] and a number of French observers that the polyhedral cells have a selective action, very much as the secreting cells of the kidney have. At times, when the cells are damaged, now one, now another ingredient is not excreted by the polyhedral cells so that there may be the presence of bile salts alone or bilirubin alone in the blood. There is not always a complete retention of all the component parts of the bile. Unfortunately the tests for the detection of the bile salts in the urine are open to criticism. However, his work is interesting and suggestive. The itching, the brachycardia, the absorption of fat, are all said to be due to bile salts. He thinks obstructive jaundice is "globale et brutale"; both salts are retained completely and suddenly. In hepatitis from various toxic substances the dissociation of the two substances found in the bile exists, now one, now the other appearing in the serum, then both appear, and again in the disappearance of the jaundice there is a similar irregularity.

The liver is, as we have said, concerned in the storage and detoxication of various substances besides bacteria. When colloidal metals are injected into the circulation they are taken up by the Kupffer cells. If the dosage is sufficient the cells are injured. The damaged function is made evident by the clinical sign of jaundice and the extent and character of the jaundice is, in a way, a measure of the impairment of the liver cells. Cunningham,[33] in studying the clinical effects of colloidal lead in patients to whom it had been administered for inoperable carcinomata, describes three forms of jaundice:

I. There is a slight icteric change in the sclera with increased urobilin and a positive indirect or delayed direct Van den Bergh reaction. The bile secreting cells are probably unaffected. The jaundice is due to excessive production of hæmolysis by the Von Kupffer cells and the reticulo-endothelial system. The pigment is produced in too large quantities to be taken up directly and secreted by the polyhedral cells of the liver parenchyma, with the result that a quantity continues to circulate in the blood.

II. There is damage to the polyhedral cells of the liver, urobilin and bile appear in the urine, the jaundice is deeper, the Van den Bergh reaction is biphasic or of direct type.

III. In the third type there is evidently a cholangitis in addition, due to the destruction of the polygonal cells. Jaundice is deeper and there are large amounts of bile in the urine. There may or may not be an increase of bilirubin of the urine; the van den Bergh test is immediate or direct.

These studies seem to me of especial interest. They furnish an indication of the way certain toxic substances act in producing jaundice by direct action on the reticulo-endothelial cells and on the polyhedral cells of the liver. We have in jaundice an obvious clinical sign and, in certain cases, an indica-

tion of the disturbance of function, either in the secreting polygonal cells of the liver or in the Kupffer cells. A similar relation of jaundice to the irritation of microbes deposited in the liver exists.

In 1916, Inada and Ido [34] and their co-workers showed the presence of spirochætæ in the blood, liver and kidneys of patients suffering from an epidemic and an endemic form of infectious jaundice occurring in Japan. They transmitted the spirochætæ by intraperitoneal injections in guinea pigs and cultivated the microparasites outside the body.

The action of the spirochætæ in producing the jaundice cannot be easily explained by obstruction of the bile passages. In the severe cases the bile passages are pervious. The lesions are confined to the hepatic cells. During the first days of the disease, before the appearance of the jaundice, the spirochætæ can be demonstrated in the blood. But the spirochætæ do not remain long in the general circulation. From the fifth to the eighth day inoculations of guinea pigs with the blood are negative; enough immunity is established to destroy the parasites in the circulating blood. The microparasites are found in the liver and other organs. Moreover, all grades of jaundice are produced. In certain cases there is no jaundice, in others it is slight and transitory, like a mild grade of catarrhal jaundice; in others it is severe and gives the picture of acute yellow atrophy. In reading the accounts of the disease it is difficult not to see a resemblance to the occasional cases of so-called acute catarrhal jaundice and there has been a general tendency during the last ten years to place fewer and fewer cases under the heading of catarrhal jaundice. The majority are assumed to be sporadic cases of infective hepatitis. Not that there is any general assumption of a spirochætal origin for all forms of epidemic jaundice. In an article by Blumer [35] in 1923, he expressly states that in the types of the disease which he has had an opportunity to see, no spirochætæ were demonstrated and that he believed that they were not the causative agents in the types usually seen in this country. But the relation of epidemic forms of jaundice to catarrhal jaundice is too striking not to assume that the ordinary forms of catarrhal jaundice are but sporadic cases of infectious jaundice, and the resemblance of the epidemic infectious jaundice is too similar to the disease produced by spirochætæ not to be noted. The clinical picture of the fatal cases, which are rare, is that of acute yellow atrophy, just as in spirochætal jaundice and here again all the evidence seems to point to hepatitis as the cause of jaundice. It is understood, of course, that acute yellow atrophy is not a disease entity but a syndrome produced by a number of toxic agents.

The autopsies which showed any lesions which corresponded to older theories of catarrhal jaundice are very few. The case reported by Eppinger [36] is of interest. A girl nineteen years old, suffering from acute catarrhal jaundice, in a fit of despondency threw herself out of a window. At autopsy the inferior extremity of the common duct was found blocked by a mass of inflamed lymphoid tissue.

The evidence to-day makes it probable that a large group of transitory

jaundices are due to damage to the liver cells, not to obstruction from catarrhal inflammation. Many microbes set up an infective hepatitis and certain of them have an elective affinity for the liver, localize there and create lesions more or less grave.

In most of the acute infections there is a period when the microparasites are found in the blood stream. Here again, after a period during which a certain degree of immunity has been established, they disappear and are found deposited in the liver and other organs. In typhoid, in pneumonia, in secondary syphilis, in malaria, etc., jaundice is occasionally present. In streptococcal sepsis and other forms of sepsis, jaundice occasionally occurs. In the fatal cases at autopsy, aside from various parenchymatous and degenerative changes, the liver shows frequently a round-celled infiltration in the interstitial tissue and focal necrosis.

Here, again, it is difficult not to believe that some degree of hepatitis is almost always present and that, now and again, either due to some special weakness, predisposition or sensitization, the parenchyma cells are enough involved to interfere with their normal function of excreting bile. The obvious clinical sign of jaundice occurring every now and again in the course of disseminated bacterial invasion, is an indication of the hepatitis which is, in all probability, frequently present and is comparable to the jaundice seen occasionally during the administration of certain toxic substances.

It is interesting to see how often transitory infective icterus appears in subjects in which the liver is already the seat of a lesion, from alcohol, syphilis or pregnancy. Possibly toxic substances set free in the violent reaction of the Kupffer cells to the multiplying microbes produce changes in the polygonal cells, disturb their function and hinder secretion. The margin of safety is so great in the liver that such hepatitis must be widespread to produce jaundice. The study of the pathogenesis of spirochætosis has transformed the significance of jaundice. It has placed a large group formerly classed as obstructive jaundice under the jaundices due to action of microorganisms on the liver cells and has called attention to the reaction of the liver cells regularly present in disseminated infection. Even in obstructive jaundice, where the main excretory ducts are closed, the liver cells are rapidly implicated. If there is obstruction of the hepatic duct above the point where the gall-bladder enters or if the common duct is completely shut off, after the gall-bladder has been removed, there may be such a disturbance of the liver cells that no bilirubin is secreted. The ducts contain whitish matter made up only of the excretions of their walls, but the failure of the liver cells to secrete bilirubin is not due to failure of the pigment to be formed. The evidence all goes to show that the bile suppression is due to a disturbance of the liver cells, and only indirectly to an obstruction to the outflow. If the gall-bladder is in place then the back pressure on the secreting cells is not so great and the damage is much less.[37] If the obstruction is intermittent, infection, either ascending or descending, inevitably follows and widespread cholangitis with further involvement of the liver cells occurs.

Various microbes not only settle out and are taken up by the liver cells in the course of disseminated infection, but reach the interstitial tissue of the liver in clumps or masses from a disintegrating, infected clot in the portal vein or by emboli through the hepatic artery, being arrested in the small vessels and producing a variety of focal lesions and multiple or single abscesses.

It has long been recognized that patients suffering from amœbic dysentery not infrequently develop liver abscesses. The amœbæ spread in the submucosa of the intestines. They may be found both in the blood-vessels and lymphatics, to be carried to the mesenteric lymph-glands or through the portal circulation to the liver, where they set up a hepatitis. The hepatitis may subside or, if the tissue resistance is poor or the dosage massive, abscesses form. Here again the liver is the first great barrier to the dissemination of the infecting agent.[38]

In many of the stages of infection when a partial immunity has been established, various forms of hepatitis occur, often giving little or no clinical evidence of their presence. According to Calmette,[39] during the course of infection with tubercle bacilli, small tuberculous lesions are nearly always present in the liver if careful enough search is made. In an autopsy reported by Sabrazes,[40] in a patient who died of leprosy, the liver showed no leprous nodules, but sections showed an enormous phagocytic reaction of the Kupffer cells. There were no bacilli, however, in the biliary passages; none in the bile. In late stages of syphilis the liver more frequently harbors treponemata than any other organ. Various forms of interstitial hepatitis, as well as the characteristic gummata, are present. Syphilis is relatively frequent. In over nine thousand Wassermann tests, taken as a routine measure in all patients treated at St. Luke's Hospital during the last year, including the out-patient department, over five per cent. showed a positive reaction. As in a number of these repeated examinations were made and as there must be a large number with syphilitic infection, yet showing a negative examination, the actual percentage should be put much higher. It would be interesting if we knew in how many of these patients there were changes in the liver, due to the lodged treponemata.

Anyone connected with a large hospital has an opportunity of seeing all these forms of hepatitis. He gets an impression of an extraordinarily complex etiology.

With these facts in mind, I shall next attempt to study the path of infection suggested in a number of recent papers and the relation of hepatitis to cholecystitis and then of cholecystitis to hepatitis.

In the first place there is a widespread notion that appendicitis, hepatitis and cholecystitis are related and that infection passes from the appendix to the liver through the portal vein. That this relation occasionally exists, in acute cases in which there is a suppurative lesion of the veins in the mesentery of the appendix and a progressive suppurative pyelophlebitis is set up, which leads to infected clots lodging in the liver, is generally recognized. But during the course of acute appendicitis, cholecystitis and hepatitis are not usually

recognized. The associated acute lesions occur, but they are not common. I do not find, in patients operated on for acute suppurative appendicitis, any disproportionate number later developing clinically recognizable forms of hepatitis. Nor do the autopsy records on patients dying of acute appendicitis as a rule show lesions of the liver. Tens of thousands of microörganisms are undoubtedly lodged in the liver, just as they are in the spleen and the bone-marrow in general sepsis from acute appendicitis. They are taken out of circulation and many of them are destroyed in these situations. Nor is the proof offered that hepatitis has been found, in removing sections of liver while performing appendectomy for chronic appendicitis, convincing. The sources of error are obviously too numerous; the impossibility of excluding other inciting agents is too great; the number of observations are too few.

Nor do I find evidence that cholecystitis regularly follows hepatitis. In the numerous instances of infective hepatitis I can find only occasional instances of involvement of the gall-bladder which reaches the grade of clinical recognition. Although hundreds of thousands of microbes are lodged in the liver there is only very exceptional evidence of acute cholecystitis. I do not question that if these patients were operated on, various slight degrees of round-celled infiltration of the wall of the gall-bladder might be found, but during the course of the jaundice that is so frequently present in infective hepatitis there is rarely present a form of cholecystitis that can be recognized clinically.

During the last year I have twice operated through a mistake in diagnosis on patients with infective hepatitis. A woman aged thirty-seven years, entered the hospital acutely ill with a temperature and jaundice. She was kept under observation. There was a history of syphilitic infection and treatment by salvarsan five months previously and, although she had had little pain, having only a sense of fulness and distention after meals, and gave no history of having had attacks suggesting cholecystitis, the erroneous conclusion was reached that she had an obstructive jaundice. On opening the abdomen the gall-bladder was full but could readily be emptied. There were no adhesions, the wall was thin, there was no dilatation of the ducts, the liver seemed a little firm. A section removed for examination showed the lesion of severe, extensive hepatitis, the lesion extending about the minute bile ducts and about the portal venules. The gall-bladder was anastomosed to the stomach. The patient made a good recovery; the jaundice very slowly subsided, as is the rule in infective jaundice. The patient, seen one year and a half later, was in good health, with no jaundice. There was marked hepatitis, proved by the microscopical section, and no appreciable cholecystitis. The second case was similar. There was hepatitis present; the gall-bladder and ducts seemed normal. The patient had an infective hepatitis imposed upon a cirrhosis of the liver.

The following case report is in point: A man aged forty-one years, entered the hospital very ill, with fever and general abdominal pain. He was slightly jaundiced. The abdomen was opened through a right rectus incision; the gall-bladder seemed normal, a

gangrenous retrocæcal appendix was discovered and removed. He grew gradually more jaundiced and died on the second day. During the last day he complained bitterly of pain in his right leg. At autopsy the liver showed marked hepatitis. The common duct was patent and there was no dilatation. The cystic duct was patent; there was no gross lesion of the gall-bladder. There was infection of the retroperitoneal tissue; the psoas muscle was soft and pultaceous. Cultures from the retroperitoneal infection showed a large non-gas-forming bacillus. This patient is an example of hepatitis following appendicitis, yet advanced acute hepatitis was unaccompanied by gross lesions of the gall-bladder.

In typhoid fever the relation of hepatitis and cholecystis has attracted much attention. The initial period of typhoid fever is accompanied by the presence of typhoid bacilli in the blood stream. After a time the bacilli are no longer found in the blood. They are found in the bone-marrow, spleen, lymphoid tissue of the intestine and lymph-glands of the mesentery. In the lymphoid tissue of the intestine they colonize, multiply and produce the characteristic lesions of the disease. The livers of patients dying of typhoid regularly show changes; there is fatty degeneration, infiltration with leucocytes, masses of leucocytes about degenerated and fragmented liver cells. These nodules are said to be due to bacterial emboli.[41] At times there is acute diffuse hepatitis or massive fatty degeneration, some of these patients are jaundiced and the jaundice, to quote Widal, is due to a hepatitis: "The jaundice of typhoid is really a hepatitis, the result of lesions of the hepatic cells, determined by the localization in the liver of microbes deposited there from the general circulation.[42] The bile regularly contains typhoid bacilli and there are all grades of lesions of the gall-bladder, from catarrhal to purulent inflammation, yet cholecystitis giving clinical symptoms is not common in typhoid fever. From the seventh to the thirtieth day pain in the right hypochondrium, tenderness and rigidity are the symptoms that all who have an opportunity to see any considerable number of patients suffering with typhoid, associate with the condition. Though thousands of micro-organisms are in the blood stream and the liver and although the gall-bladder wall frequently shows lesions, only occasionally do symptoms appear that bring the lesions into the plane of clinical observation. Other factors seem to be necessary. One would expect that occasionally bacterial emboli might be lodged in the wall of the gall-bladder. One would expect that occasionally bacteria would be carried by the lymph stream to the wall, but it is difficult not to believe that the thousands of organisms in the infected bile must play a most important part in producing lesions of the gall-bladder. The experiments of Nichols are suggestive.[43] He succeeded in 63 per cent. in producing gall-bladder lesions when injecting typhoid bacilli by the mesenteric vein and in 41 per cent. when injecting into the systemic veins. There was a considerably higher percentage of gall-bladder infections occurring in the immune animals than in the normal animals. He brought forward the view that gall-bladder infections in inoculated animals are not necessarily an index of immunity, but may be in part an indication of a rich amount of immune bodies in the blood.

The colon bacillus, aside from infection of the liver following stones lodged in the common duct and consequent infective cholangitis, can, in exactly similar manner, cause jaundice. The following autopsy report is an example: The skin was jaundiced, the liver showed acute hepatitis, the common duct was normal in appearance, the gall-bladder showed a subacute lesion. There was extensive degeneration of the liver cells and central necrosis, yet the gall-bladder showed only a subacute lesion. Cultures taken from the gall-bladder bile showed colon bacilli.

In the purulent form of interstitial hepatitis it is curious to see the absence of correspondence in the lesions of a gall-bladder and the lesions of the liver. A child, ten years old, very ill, was admitted to the hospital. She had a high temperature, tenderness and an indistinct feeling of mass in the right hypochondrium. The abdomen was opened, the liver was found enlarged, the gall-bladder seemed acutely inflamed. It was removed. The patient died in three days. At autopsy there was only a small amount of serous fluid in the upper part of the abdomen. There were no signs of peritonitis. The liver was enlarged and riddled with abscesses. There was an early parietal clot forming in the portal veins. Section of the gall-bladder removed at operation showed a subacute cholecystitis. Although the liver was filled with abscesses, some of them but a few centimetres from the gall-bladder, only enough bacteria had passed through the lymphatics to set up a subacute lesion. Cultures from the pus in the abscesses showed colon bacilli.

In amœbic abscesses I have been unable to find records of gall-bladder involvement. Although tubercular lesions of the liver are said to be common, tuberculosis of the gall-bladder wall is rare. Simmonds [44] has given a description of the forms. In syphilis, although various forms of hepatitis are common, there is no evidence of unusual frequency of clinical forms of cholecystitis. As I understand the evidence, there is no unusual incident of gall-bladder infection following the various forms of hepatitis. Bacteria are probably not infrequently present and not infrequently there are slight evidences of an inflammatory reaction. To produce a clinical form of cholecystitis, added factors are necessary.

If the gall-bladder is grossly infected, if the bacteria are lodged in its wall and the tissues react and the lumen is filled with exudate and bile, does a form of hepatitis develop, leading to clinical forms of cirrhosis? A reacting zone of contiguous structures is formed about the inflamed organ. The peritoneum, the wall of the colon and the duodenum are glued to the gall-bladder by exudate. The omentum turns up and is adherent to the gall-bladder and the edge of the liver; the cellular tissue between the gall-bladder and the liver reacts and the interstitial tissue of the liver reacts. The extent of the zone of circumscribing reaction depends, as in all infections, on the virulence of the lodged organisms and the local and general resistance. There are all grades of severity, from inflammatory changes that can hardly be detected by microscopical study, to changes where purulent exudate and ulceration and necrotic processes are the characteristic features; and in all

of them the liver in contact with the gall-bladder shows varying grades of hepatitis. Moreover, if the attacks are repeated there may be evidences of chronic interstitial inflammation. Sections taken of the quadrate lobe or the right lobe in the neighborhood of the infected gall-bladder regularly show signs of inflammation on microscopical examination.

Anyone who has operated on a number of gall-bladders recognizes, as a matter of course, the infiltration of the cellular tissue between the gall-bladder and the liver, succulent in fresh infection, dense and firm in long-standing cases. When the gall-bladder ulcerates or is necrotic at some point an abscess may form partially in the liver substance. There can be no question that some form of local hepatitis occurs in almost all infections of the gall-bladder and that the infection travels from the gall-bladder to the liver. It is a matter so generally accepted that it has rarely been emphasized. It is part of the pericholecystitis nearly always present. Specimens, however, have been taken in many instances far enough away from the bed of the gall-bladder to indicate that the reaction is widespread. There is no means of knowing in these instances whether the reaction in the liver may not be due to one of the many irritants which reach the liver. This may readily be the case if the gall-bladder lesion does not present gross morphological changes. In any event, the hepatitis which we see so frequently at operations for cholecystitis does not seem to have clinical significance, for if the grossly inflamed gall-bladder is removed there is no progressive involvement of the liver. In several cases in which I have removed a gall-bladder with an abscess between the gall-bladder and the liver, the infection in the liver has apparently promptly subsided, for these patients are in good health and fat when seen years after their operations.

A patient entered the hospital two years ago presenting several interesting features. He had a bottle in his hand containing gall-stones that he had passed twenty years ago. He had had, after this long period of latency, a severe attack of typical gall-bladder pain. Twenty-seven years ago he had had his first attack and during seven years following had had a number, several of them with jaundice. During the last one of these attacks he passed the stones he showed. He was operated on by Doctor Bolling and an obviously inflamed gall-bladder was removed, containing stones in every way similar to the ones he had passed many years before. The liver seemed firm and the edge a little rounded. The wall of the gall-bladder showed a small carcinoma. He had at that time and still has, two years later, no signs of chronic hepatitis or liver cirrhosis that can be appreciated clinically, although there had been enough irritation to produce a new growth, and although the irritating agent had acted over a period of twenty-seven years.

There is another bit of evidence worth recording. Fifteen years ago, in a number of the large clinics, the gall-bladders were not removed. The stones were removed and the gall-bladder drained. The practice was abandoned. There were too many recurrences due to impacted stones or stenosis of the cystic duct. Many of these patients recovered. I can recall no instance

or report of the retained, grossly infected gall-bladder setting up chronic forms of hepatitis. I gained the impression at that time that a draining gall-bladder, even if infected, gave little or no symptoms.

In deeply jaundiced patients with septic fever and a stone in the common duct, I have in several instances left the shrunken gall-bladder in place, opened the common duct and removed the stones. In no instance has a progressive hepatitis or liver cirrhosis followed.

I do not find evidence, then, of a so-called vicious circle; a chronic appendicitis, then a hepatitis, then a cholecystitis and the cholecystitis then becoming the main focus and producing hepatitis. The evidence seems to be that the liver is a most efficient filter for bacteria, taking them up from the general or portal circulation and that only a few pass the cells of the peculiar liver capillaries to reach the lymph and float along with the lymph current into the nodes at the hilus. At autopsy or at operation these nodes are not infrequently enlarged, but the enlargement of the lymphatic nodes is an inconspicuous feature of infective hepatitis. I do not question that a few bacteria may pass along the lymphatics from the liver into the wall of the gall-bladder. I do question, however, whether slight infiltration of the gall-bladder wall, changes so slight that it is impossible to recognize them by gross examination, are clinical forms of cholecystitis. This assumption seems to be based on the observation that certain patients gain relief from symptoms of a form of dyspepsia after the removal of a gall-bladder which shows no macroscopic lesions or shows beneath the mucosa, when cut open, small yellow spots from lipoid deposits (strawberry gall-bladder). This form of indigestion is shown by accumulations of gas in the upper abdomen, belching, sour regurgitations occurring promptly after eating a hearty meal or badly prepared or indigestible food.[49] Unfortunately these symptoms are present to some degree in most middle-aged people. They are present, as I have assured myself, at times after the gall-bladder has been removed and they are absent in many instances in which there is a gross lesion of the gall-bladder wall.

In the same way study of the cholecystograms, made by the method recently introduced by Graham, has been said to furnish a surer means of recognizing gall-bladder disease than the sight and touch of the surgeon at operation; that is, by the observation of the filling and emptying of the gall-bladder one can determine whether a gall-bladder is so diseased that it should be removed, even if there is no demonstrable lesion at operation. The gall-bladder disease is proved by the microscopic examination of the excised gall-bladder wall, showing a very slight cellular infiltration and by statements made by the patients of relief after operation. But is it not too early to draw this conclusion; is not too little known of the normal function to speak so confidently of abnormal functions? I grant that certain patients get complete relief after operations for the removal of the gall-bladder with very trifling lesions of the gall-bladder wall, but a more or less lasting freedom from symptoms has been presented as proof for all manner of remedies. Patients treated by homeopathy and osteopathy are sincere in their belief

in the remedies applied and grateful for the relief afforded. I believe we should demand stronger proof. I have an instinctive distrust of occult surgery or surgery for occult lesions. I recently saw a middle-aged woman operated on in a large clinic. Her symptoms were, as nearly as I can remember, gas, indefinite gastric discomfort, inability to eat certain foods, and pain after eating. When the gall-bladder was exposed it was soft, thin-walled, on manipulation it emptied, there were no adhesions, no stones. The gall-bladder takes up and concentrates bile and regulates the pressure in the biliary system.[47] If the orifice of the cystic duct is blocked, there is evidence of disturbance and pain. The gall-bladder in question emptied, it was thin-walled and distensible. It contained stasis bile. Which of its known functions was at fault? The gall-bladder was removed, it was cut open and from the appearance of the thick, stringy stasis bile, cholecystitis was said to be present.

The pathologist, Aschoff,[48] whose painstaking and thorough studies have thrown so much light on the formation of gall-stone and the function and structure of the extrahepatic bile passages, has pointed out that in studying post-mortem staining of the biliary passages there are fairly constant findings. The region of the sphincter of Oddi is usually pale, almost colorless, a sign that it is closed, even after death. The common and hepatic ducts and the cystic duct up to the neck of the gall-bladder are stained yellow, that is, stained with hepatic bile, while the neck and the rest of the gall-bladder are stained dark brown by the stasis bile of the gall-bladder as if the change in color marked a division between the conducting system and the condensing system of the extrahepatic bile passages. He also points out that there is sphincter-like thickening of the smooth muscles at the beginning of the cystic duct and numerous ganglion cells and nerve fibres in the wall of the cystic duct. We apparently have to think not only of the mechanism of control at the orifice of the common duct, but of a mechanism of control at the outlet of the gall-bladder. That among the patients in whom the gall-bladder has been removed for trifling lesions and in whom the cysticus is patent, there are a few who have had definite cramp-like attacks due to disturbance of the nervous control mechanism which synchronizes the various sphincter-like structures at the orifice of the cystic and at the orifice of the common duct, seems very probable. But in the great majority of cases, to produce a clinical form of cholecystitis, either very unusual virulence of the microörganisms is necessary or there is added to the factor of slight infection a disturbance either mechanical (calculus, anatomical peculiarities, etc.), or functional, which interferes with the passage of the gall-bladder contents through the cystic duct.

TO SUMMARIZE

I. One of the main functions of the liver is the destruction and disposal of bacteria and toxic substances.

II. Bacteria and toxic material reach the liver in a great number of ways and very frequently throughout life.

III. Some are destroyed there without appreciable reaction and all grades of appreciable reaction occur.

IV. The morphological changes which occur in the liver in the reaction of the cells to irritants and which are called by the pathologist hepatitis are extremely difficult to classify. The subject of hepatitis is one of the most complex in the domain of pathology. It is in no sense an entity nor can it be discussed with profit as an entity.

V. Many of the cases formerly grouped under acute catarrhal jaundice are now considered forms of infectious hepatitis.

VI. In the forms of hepatitis which have reached the level of clinical observation there is little evidence of a relation to clinical forms of cholecystitis.

VII. The hepatitis regularly found with cholecystitis has little or no clinical significance and is not a factor of importance in causing clinical forms of liver cirrhosis.

VIII. The proof that very slight infiltration of the gall-bladder wall and lipoid deposit in the mucosa cause acid indigestion, a feeling of fulness in the epigastrium, flatulence, intermittent gastric pain after eating badly prepared food, is by no means conclusive.

In closing, may I quote the following letter of advice to a young noblewoman from Burton's *Anatomy of Melancholy:* "In this hypochondriacal or flatuous melancholy the symptoms are so ambiguous that the most exquisite physicians cannot determine of the part affected." The symptoms of this disease are said to be "sharp belchings, fulsome crudities, heat in the bowels, wind and rumbling in the guts, vehement gripings, pain in the belly and stomach sometimes, after meat that is hard of concoction." I am not sure whether Burton's rendering is better than the original Latin, which I add: "Acidi ructus, cruditates, æstus in præcordiis, flatus, interdum ventriculi dolores vehementes, sumptoque cibo concoctu difficili."

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PRE-OPERATIVE AND POST-OPERATIVE TREATMENT OF THE GALL-BLADDER PATIENT

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In scanning the history of gall-duct surgery one is impressed by the influence of simple mechanics upon the early period, and the complex chemical problems being worked out in conjunction with the surgery of to-day. The earliest period, or stone age, produced a type of surgery which had for its purpose removal of an obstruction to the cystic or common duct. Cholecystostomy and choledochotomy were practiced for the removal of stones without due consideration for recurrence, or the effect upon the liver and adjoining organs of the diseased gall-bladder which remained. The second period, or local infection age, produced a type of surgery planned for the removal of stones and prevention of their recurrence. During this period the local infectious influence of a diseased gall-bladder was revealed. The last and present period, or chemical age, although not completely developed, demands that the surgeon in planning operative procedures upon the gall-ducts include all those principles of the earlier periods with the changes in internal metabolism which have taken place as a result of disease of the liver and gall-ducts. Another factor of this period is the influence upon the liver and its function of infectious disease in other organs.

The preparation for operation and the after-care during the first and second periods were not specific for patients with liver disease. The general measures in use at that time were developed from the experience of surgeons in operations of all kinds. Acute infection as cholecystitis and cholangitis played a specific part in this instance in the preparation of the patient by delaying the operation for the infection to become encysted and attenuated in virulence. Hindsight, together with the chemical knowledge of liver function of to-day, leads one to believe that the rôle infection played in the outcome was over-emphasized, and recent results of operations in the presence of acute infections seem to bear this out. In the presence of a normal metabolism delay in operating upon patients with acute cholecystitis or cholangitis is not necessary, and chemical changes in the circulating tissues of the body indicate it to be injurious.

Careful consideration of the specific factors in the preparation and after-care of individuals with gall-duct disease will be the means toward a further reduction in the mortality of the operative period. There are certain definite facts to be determined before and after operation which will designate the course of action in preparing the individual for so strenuous a period, maintaining a supply of suitable raw products of metabolism, and avoiding the dangers from the disordered system of liver metabolism. The chemical labora-

tory is a necessary constituent of every hospital, and in this instance, determinations made are of great assistance to the surgeon in the care of the patient and in increasing the knowledge of liver metabolism.

Acidosis.—Changes in metabolism and the occurrence of an acidosis are seldom due to destruction of liver substance and loss of function but occur from the disturbance in food intake due to the secondary effect of the diseased gall-bladder upon the stomach. In the acute gall-bladder vomiting of food occurs repeatedly during the course of the infection. After twenty-four hours the reserve supply of glycogen, which may have been normal, has been used up and unless carbohydrate is furnished in some form an acidosis surely occurs. By the aid of blood examinations the presence and extent of acidosis may be determined and corrected. Before operating upon a patient, acutely ill for thirty-six hours, the CO^2 combining power should be determined. When it is found to be below forty, measures for relief should be instituted and the operation delayed. A mild acidosis of this type can be corrected by the administration subcutaneously of 1500 c.c. of glucose 3 per cent. and normal saline. To operate upon a patient with a CO^2 of forty is perfectly safe if it remained forty, but in every instance, following the trauma and anæsthetic there will be a further reduction which makes the after-care more difficult. A CO^2 of thirty should be corrected by the administration subcutaneously of 1000 c.c. glucose 3 per cent. in normal saline, intravenously 1000 c.c. of 3 per cent. glucose and by rectum 10 per cent. in ounces six, every four hours. At the end of six hours another CO^2 should be done and if the rise has not been to forty-five or above, the administration should be repeated. Instances of CO^2 below thirty will require in addition to the glucose and chloride, soda bicarbonate 1 per cent. added to the intravenous solution and 2 per cent. to the rectal solution.

Soda bicarbonate in more concentrated solution or more than ten grains should never be given before taking the CO^2. If the acidosis does not respond the Ph. should be done before increasing the amount.

Glucose in concentrated solutions of 5 and 10 per cent. is a marked diuretic producing an increase in viscosity of the blood. Also the glucose is lost by way of the kidneys when given in concentrated solutions. When time permits or the amount desired be moderate the subcutaneous administration of 3 per cent. solutions is the method of choice.

In chronic gall-bladder disease the intake of carbohydrate is not as a rule interfered with. The fats are the disturbing element of the food and such patients come to operation well prepared for the acute operative metabolic emergency.

In external gall fistula of long standing the metabolism varies. The drainage of bile salts does not produce a constant acidosis and in these patients preparation should be controlled by chemical analysis of the blood. No measures for relief of an acidosis should be employed until it has been determined to be present.

Without chemical analysis of the blood, no patient should have soda bicar-

bonate intravenously. Large amounts of acetone and diacetic acid in the urine may occur in patients with an alkalosis. When acetone and diacetic acid appear in the urine and the patient presents other symptoms of acidosis,

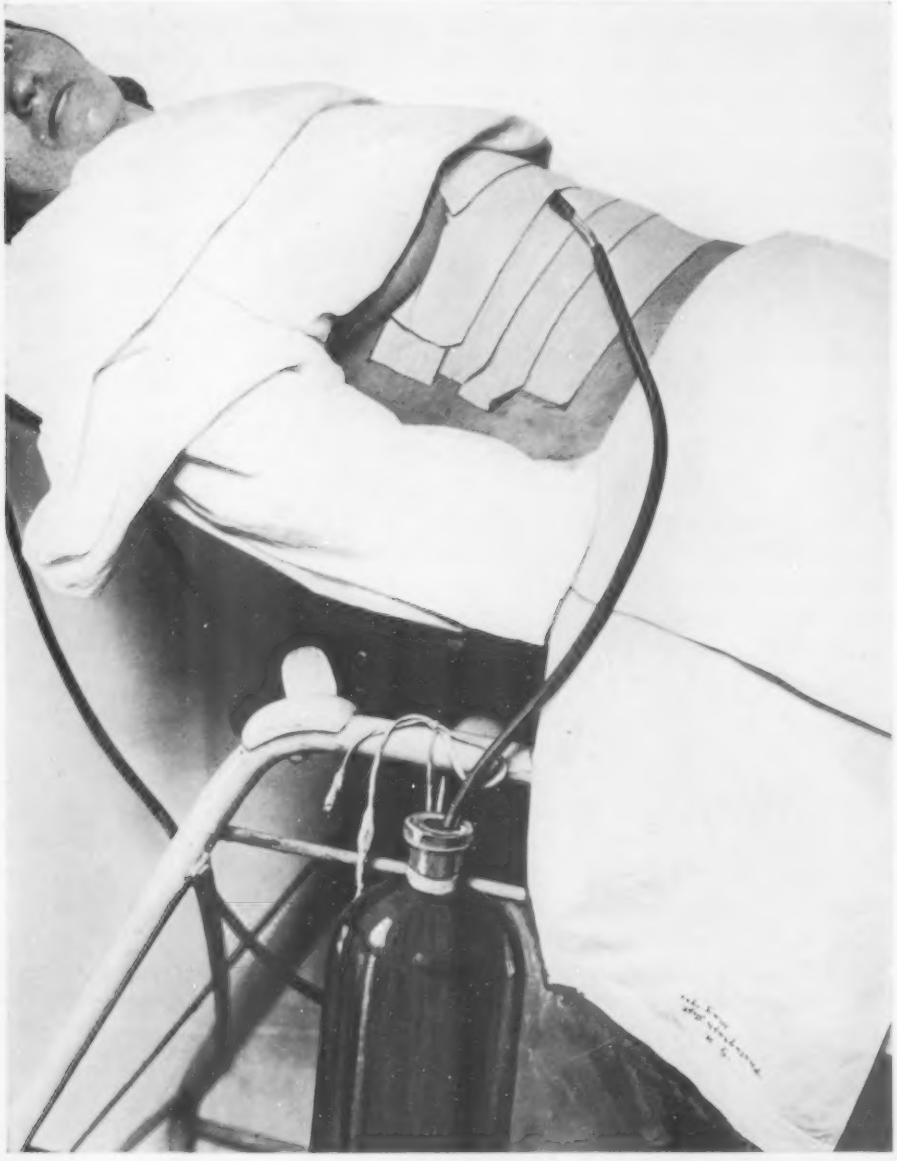

FIG. 1.

glucose and normal saline solution should be used until the urine is free of these incomplete products of fat metabolism. When the patient has starved for thirty-six hours and urinalysis cannot be had, normal saline in amounts consistent with the period of starvation, condition of the heart and general

appearance of the patient is indicated. Periods of starvation over thirty-six hours in a patient with a history of diabetes should have glucose added in 3 per cent. amounts to the saline solution. To give a patient with acidosis and a high blood sugar concentrated solutions of glucose causes a depletion of body fluids in excess of the amounts introduced, and as these patients run a high viscosity they are in danger of coma following further depletion of fluid from the diuretic action of the glucose.

Alkalosis.—A state of alkalosis existing even in mild form before operation is rarely encountered. Only in those patients with an accompanying ulcer, usually duodenal in type, who have developed the soda bicarbonate habit is it apt to be present. In such individuals the taking of soda bicarbonate should be discontinued one week before operation in chronic disease, and in the acute, a chemical blood analysis made. In the latter instance a CO^2 above seventy-five should cause the operation to be postponed in all but those with ruptured viscus or extremely high temperature with chills, indicating a severe cholangitis. When the operation can be delayed the patient should have dilute Hcl. minims fifteen in water, ounces six, every three hours by mouth, and dilute Hcl. minims twenty in water, ounces six, every six hours by rectum; and the subcutaneous administration of 3 per cent. glucose in normal saline 1500 c.c. in twenty-four hours should be used. In extreme instances of the CO^2 being above eighty-five operation is fatal unless vigorous treatment be instituted. Unusual instances of this sort are always discovered post-operatively and will be discussed later.

Jaundice.—An objective symptom was as much a problem during the stone age as it is to-day. However, the control of the bleeding which occurs in those jaundiced from obstruction to the common bile ducts has been completely and simply carried out during the present period by means of the intravenous use of calcium chloride. During the second period of gall-duct surgery, blood transfusion was used for this purpose and it was successful. Blood transfusion, however, is expensive and is not warranted for the control of bleeding alone. In debilitated patients who also have an increase in clotting time, whole blood remains the best means of preventing or controlling hemorrhage. Calcium chloride which is put up in convenient ampules, in 10 per cent. solution should be administered intravenously in those having a clotting time of over seven minutes, 10 c.c. given every six hours until the clotting time has been reduced to normal or for three injections.

Post-operative Feedings.—In all instances of operations outside the gastro-intestinal tract feedings should be resumed after eighteen hours. In patients with simple cholecystectomy, fluid as tap water should be resumed after six hours in amounts sufficient to satisfy thirst. Food in the form of gruels, dry toast, cooked fruits and sugar dissolved in drinks of tea, orange phosphate and pineapple phosphate should be insisted upon after eighteen hours. No acids, either as orange, grape or lemon juice should be allowed, neither ice water nor cracked ice.

In the acute case fluids by mouth will not be tolerated well before twelve

hours, and rentention enemata of water ounces six with glucose ounces one should be given every four hours during the first day. Then unless there are evident signs of a spreading peritonitis the feedings of starch and sugar by mouth should be insisted upon. Gaseous distention, if not due to operative trauma or peritonitis is best relieved on the second day by feedings.

Position.—In the simple cholecystectomy any posture the patient may prefer is permissible. When drainage of the common duct or abdominal drainage of an acute infection is present the sitting posture of thirty-five degrees should be used to promote drainage and prevent subphrenic collections of bile or purulent material.

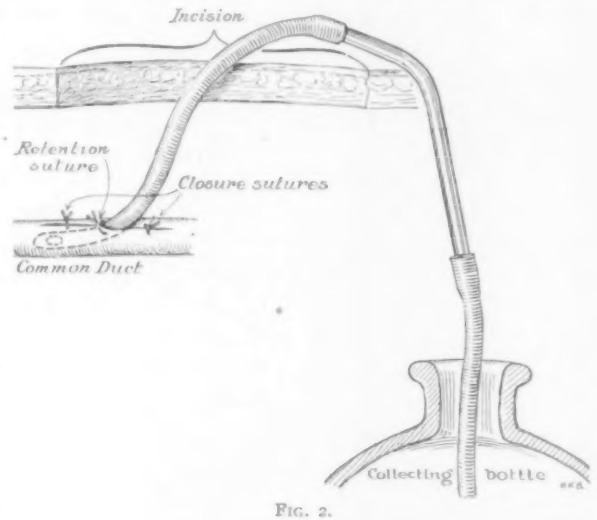

FIG. 2.

Pain.—There is one characteristic type, spasmodic, breath taking and alarming. Soon after regaining consciousness, the patient complains at intervals of a spasm which grips the epigastric region and often is referred to the back and shoulder. In all instances of this sort, I have noted later drainage from the wound of bile or broken down blood-clot. It is not caused by lack of drainage of the common duct for patients with severed common duct complain, if at all, of a continuous pressure somewhere in the upper abdomen. Morphine in large amounts will not entirely relieve the suffering and will not stop the spasm. If due to intra-abdominal drainage of bile the drainage should be loosened or the sutures adjoining the drain cut. If due to intra-abdominal hemorrhage, morphine in large doses to relieve the pain which will persist for eighteen hours usually suffices and at the end of five days broken down blood clot will be extruded to explain the cause of the spasm and pain. There is an expiratory grunt similar to that accompanying pleurisy and explanation of the pain may be that the retained material is forced up under the diaphragm.

Hemorrhage.—There are two types peculiar to disease of the liver and ducts. First, the ooze from the fine capillaries due to changes in blood coagulation. At present and only in acute instances will this form be encountered for the preliminary use of calcium chloride and blood transfusion have eliminated this dreaded type of bleeding. When in the acute or neglected instances of its occurrence, calcium chloride should be administered in 10 c.c. of 10 per cent. intravenously every four hours until the coagulation period returns to normal. Should this means fail and if there has been considerable

blood loss to hæmoglobin of sixty or below, blood transfusion should be resorted to.

Hemorrhage from within the common duct with the ulceration of a liver sinus in ulcerative cholangitis may be sudden and profuse. Morphine in large doses and transfusion to replace the blood loss are indicated. This type of hemorrhage is rare and when it occurs is usually the terminal stage of a prolonged liver infection.

Common Duct Drainage.—Drainage tubes introduced into the common duct for external drainage are usually sewn in place. Instances of broken tubes with a portion left in the duct necessitating choledochtomy have been reported. Any pull exerted upon the common duct tube is to be very carefully avoided. Extending the tube by means of a connecting link which carries the bile to a bottle fixed to the bed railing is a common practice. (Fig. 1.)

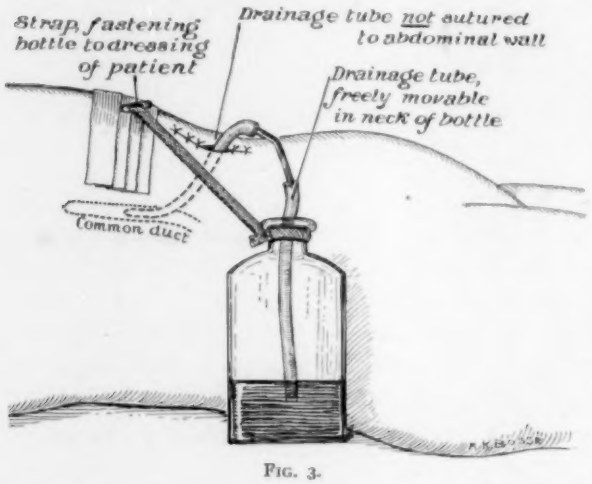

Fig. 3.

As this tube enters the bottle, usually one with a narrow neck, at a perpendicular plane, it will bind and catch if withdrawn at an angle. In turning the patient the tube is pulled upon at an angle which often results in binding at the bottle neck, with pull being put upon the other end which is firmly sutured to the common duct. (Fig. 2.)

The stitch in the common duct may tear out leaving a transverse tear which may later result in an obstructing cicatrix, or the tube may break at its weakest point up to the stitch and if the weakest point, as it usually is, be at the stitch, the tube will come away leaving a portion in the common duct. To avoid the possibility of breaking off the tube in this way, Dr. John F. Erdmann has devised the plan presented. The drainage tube is connected by means of an angle glass tube with a section of tube long enough to extend to the level of the bed. Around this second length of tubing, and not attached to it in any way, an ordinary eight ounce specimen bottle is placed. And the bottle is attached to the adhesive dressing, or tie straps, by looping one end of a length of tape around the neck of the bottle and pinning the other. (Figs. 3 and 4.)

This arrangement prevents traction being put upon the tube, and also kinking of the tube, which may occur when long tubes are used. Turning the patient is facilitated by this means, in that the bottle, which carries the tube, is merely raised and carried to the opposite side by the attendant or by

the patient. Little care is needed to prevent spilling the contents of the bottle, when the tape is made only long enough to permit the bottle to rest upright on the bed. (Fig. 4.)

Acidosis.—Post-operative acidosis when mild usually presents on the second day. It may be from either intensive preliminary catharsis, starvation for two or three days in preparation for operation or starvation because of the pain and nausea attendant upon the passage of stone. Obese women

FIG. 4.

are most often affected and in such apparently well-nourished individuals it is at first a surprise. The storage of sugar is not greater in the obese than in the thin individuals and need of preparation of carbohydrate is less apt to be apparent in the fat than in the lean. The appearance is characteristic. In the afternoon of the second day the patient becomes restless, face flushed, complaining of generalized pain and of feeling miserable, desires cracked ice, will take ice water or orange juice in large amounts. Vomiting of stomach and duodenal contents occurs, distention is marked and not relieved by enemata, the tongue is dry and parched, lips covered with scaling epithelium. There is a rise in pulse, temperature and respiration. The pulse is good and the temperature seldom rises above 103 degrees, the respiration is fast

and shallow. Orange juice is the desire of the majority and the most injurious drink in that the hyperacidity and gastric distress are increased. Treatment of this type does not require chemical blood analysis. Sugar and water are required. Administration differs, by rectum normal saline ounces six and glucose ounces one every three hours is the mainstay—water of a moderate temperature by mouth in regulated amounts of ounces three every hour. No ice either externally or internally should be allowed. After twelve hours gruel, dry toast, tea with sugar and vegetable broth thickened with starch must be insisted upon. A firm binder for distention after a medicated enema with the frequent use of the rectal tube. For restlessness which is due to lack of fluid, besides the fluid, codein and bromide suffice to quiet the patient until the fluid loss has been replaced. No soda bicarbonate either administered to be retained nor in auto lavage solution should be permitted. For while the clinical difference between acidosis and alkalosis is fairly well established, some patients with typical symptoms of acidosis may in reality be in the state of early alkalosis. In which case: glucose, normal saline and water are indicated and of general value, while soda bicarbonate is fatal.

The severe type of acidosis occurs in acute infections of long standing and usually in company with a post-operative spreading peritonitis. The picture is different. There is a general expression of horror which is characteristic, the eyes are sunken, white showing, rimmed with dark moist lids. The mouth is open, sordes covered teeth appear before a tongue dry, parched and bile-stained over which the moist acetone breath is drawn rapidly back and forth in quick gasps which are frequently augmented by deep sighs ending in moans of despair much more impressive than those arising from acute localized pain. The skin is moist, dusky and lifeless. The pulse races in small volume and is the true thready variety. Generally there is, next to that from hemorrhage, the most impressive type of restlessness, both due to air hunger. This is the danger stage and one requiring laboratory aid for its relief. Blood analysis to determine the CO^2 combining power, urea, chloride and sugar is imperative. Preliminary measures are those indicated in all instances of fluid loss, namely: glucose, water and sodium chloride. Subcutaneous administration of glucose 3 per cent. in normal saline 1000 c.c., intravenous medication except in specific amounts is not indicated nor well tolerated by these patients. After determining the CO^2 which should not require more than one hour, should it be below thirty, intravenous glucose 3 per cent., soda bicarbonate 1 per cent. solution 1000 c.c. should be administered. In a quick response within one hour the patient should show improvement and will if the peritonitis be not too extensive or virulent in character. Repeating the subcutaneous glucose in normal saline in eight hours will restore the patient to the point where retention enemata of glucose and normal saline will suffice. Carbohydrate by mouth should be withheld for a period of eight hours, after last vomiting. Beginning with tea and sugar and increasing to gruel, toast and vegetable broths. No patient with an acidosis and a CO^2 combining power

of over fifteen in the absence of heart or kidney disease should be considered hopeless or even a difficult problem.

Blood analysis repeated every twelve hours is a necessity in the well executed treatment. To proceed blindly to give soda bicarbonate as was done five years ago will in many instances convert the picture from a simple acidosis into an irreparable alkalosis.

Alkalosis.—Unfortunately for the patient and one in attendance, alkalosis

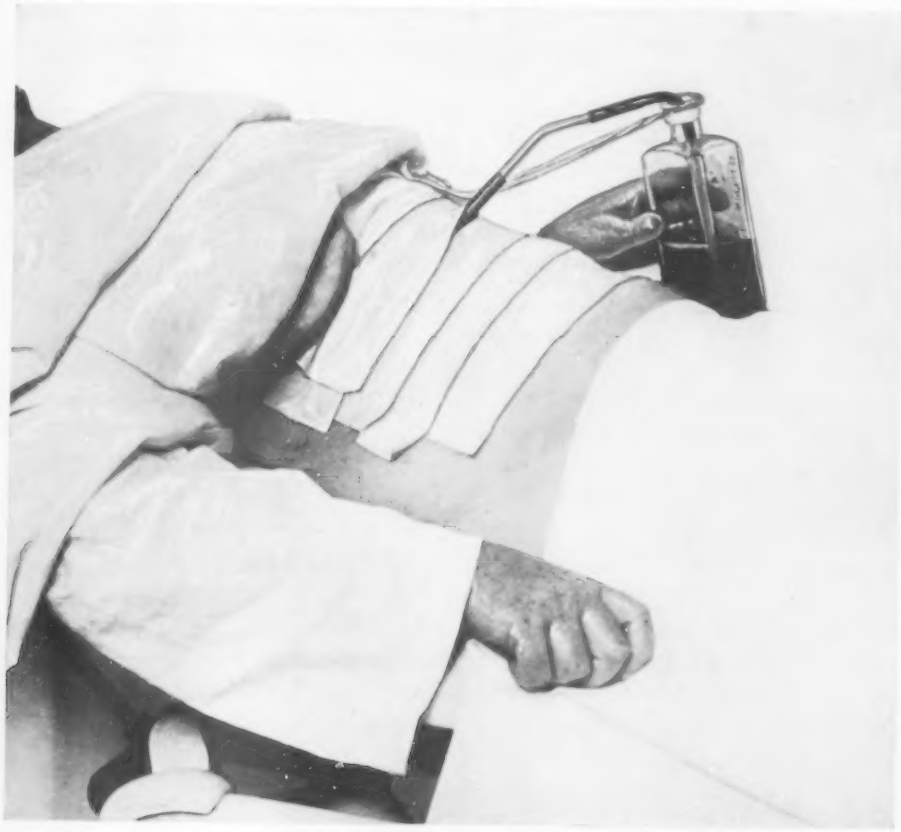

FIG. 5.

is generally discovered late. Of subjective symptoms there are none and the objective cannot be divided into early and late periods. One should be on constant guard and then late discovery will be the rule with the present knowledge on this subject. Prodromal symptoms are not characteristic but in the presence of biliary drainage, a complaint of weakness, lethargy and a rise in temperature and scant drainage of precipitated bile, a CO^2 determination should be done. In many instances normal findings will be followed by a good night's sleep and a bright and cheerful patient in the morning. In too many a quiet night, rise of temperature, cessation of biliary drainage and a stuporous patient ensue. So the danger point is reached and past before one is well aware of anything alarming.

The well developed alkalosis transforms an individual from a state of quiet composure to one of quiet tension or twitching somnolence. The facial expression remains normal for deep slumber, eyes and mouth closed; slow, sonorous respiration; the extremities are held in tonic spasm with the knees flexed upon the trunk. The tongue is moist and the eyes rolled up, pupils normal. There may be a carpo pedal spasm, however, many die without developing the typical symptom. There may be twitching or a coarse tremor of the extremities. The skin feels normal; and only as a terminal sign do the temperature and pulse rate go up to very high levels, twelve to eighteen hours before death.

Treatment presents a peculiarity noted in these patients in that Hcl will do more toward recovery than in alkalosis of other conditions. The most severe type may occur in individuals who have not vomited and in those without drainage of bile. There is no rule of occurrence but the dependable relief is in the administration of Hcl in large amounts. By mouth fifteen minims in water ounces six every two hours. By rectum twenty minims in water ounces six every three hours. In severe instances calcium chloride by vein 30 c.c. of 10 per cent. solution in divided doses can be used. Large infusions of normal saline and glucose are contra-indicated from experience and there is not the drop in chlorides of the blood as noted in alkalosis of intestinal obstruction.

Residual Infections.—Foci of infections have been ridden extensively through the literature on all subjects. But assuredly there is a permanent place in the after-care of gall-bladder patients for this subject. Infected teeth, tonsils, nasal sinuses and within the abdomen diseased adnexia, uterus, cervix and colitis are a cause for recurrent biliary explosions. Following a flare-up in any of the above mentioned foci there occurs, a pseudo gall-bladder attack as characteristic of a primary gall-bladder involvement as if the gall-bladder were present. Relief from recessions of this sort does not come from food control but from radical attention to the existing focus of infection.

SOME RESULTS OF SURGERY OF THE BILIARY TRACT*

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and
L. K. Ferguson, M.D.
of Philadelphia, Pa.

This paper represents a study of 136 cases of biliary tract disease. The cases are all personal ones, most of which were operated upon while on Service C of the University of Pennsylvania Hospital. The review includes 33 operations for acute gall-bladder disease with 2 deaths, 78 operations for chronic gall-bladder disease with 2 deaths, 21 operations for biliary disease with obstructive jaundice with 4 deaths, and 2 operations for carcinoma of the gall-bladder with 1 death, and 2 cases with associated duodenal ulcer with 1 death. A follow-up study has been made and a response has been obtained from 94 per cent. of the patients.

In an effort to clarify or confirm the existing ideas concerning the pathogenesis of gall-bladder disease, a study was made with respect to sex, age, and race, obesity and the relation of the disease to pregnancy.

Race and Sex.—Of the 134 patients with gall-bladder disease 83 were females and 17 were males. There are 3 negroes in this series. This relative infrequency of biliary disease has been noted by many authors. It would seem, therefore, that comparative studies between the white and black races might do much toward coming to a final definite decision with regard to the etiology of gall-bladder disease.

Age.—Less than 20	20–29	30–39	40–49	50–59	60–69	70+
No. of cases 1	12	33	37	30	12	3

The group between twenty and forty years, usually considered below the gall-bladder age, included 36.5 per cent. of our patients. Sixty-five per cent. of the cases in females came to operation during the child-bearing period.

Obesity.—A note designating the patient as obese was found in 76 cases. In all but 2 of the cases occurring in males, obesity was found.

Relation to Pregnancy.—Some interesting observations were made of the relation of cholelithiasis to pregnancy. The data was not complete in all cases, but using that which was available it was found that 74 of the 111 female patients had passed through one or more pregnancies. Twenty-two of these noted that their attacks occurred or recurred during or shortly after a pregnancy. One patient gave a history of having an attack of gall-stone colic follow each one of five deliveries. A large majority of our cases in women between twenty and thirty occurred during or immediately following the first or second pregnancy.

With these factors in mind, we may say, then, that cholelithiasis is a disease primarily of women, that it occurs most often in the obese, and that it

* Read before the Philadelphia Academy of Surgery, December 6, 1926.

bears a definite relation to pregnancy. We believe then that gall-stone formation is not primarily a phenomenon of infection, but that it is a metabolic phenomenon brought about by an effort on the part of the body to excrete an increase of cholesterol from the blood. Any condition which causes an increase of cholesterol in the blood over a considerable period of time would predispose to gall-stone formation. It has long been known that during pregnancy there is a distinct hypercholesteræmia, and, since the blood cholesterol parallels very closely the blood fats, it is not hard to believe that a hypercholesteræmia had occurred in a patient who has covered her body with a thick layer of fat.

No doubt infection plays a very prominent part in gall-bladder disease, but we believe its rôle is late in the process. The operations performed on young women disclosed gall-bladders which were normal to inspection and to palpation. They contained single or multiple stones which were soft, round, and slightly lobulated in appearance. They were composed of almost pure cholesterol. The bile, which was cultured in several cases, was sterile. These cases were looked upon as the earliest form of gall-bladder disease.

The patients showing evidence of infection were found in the older patients with thickened gall-bladders and facetted stones, indicating a disease which had probably been present for years. It is the long-standing irritation of the gall-bladder wall by the presence of stones and the trauma caused by distention when a stone obstructs its outlet which open the way for infection by bacteria which are constantly being excreted through the bile from the liver or which come to it from the blood stream.

Principles of Treatment.—Few, if any, cases of gall-bladder disease need be considered surgical emergencies, and the disease is serious enough to demand thorough pre-operative study and treatment. The only exceptions to this dictum may be found in the acute group and even among this number are many which would be benefited rather than harmed by a day or two of observation.

The pre-operative treatment may be divided into two parts. First, measures for the relief of pain, of which hot poultices of flaxseed applied to the whole abdomen, continuously, seem the most efficacious. If necessary, morphine may be given hypodermically in addition. The patient should be given nothing by mouth. By this means the vomiting is controlled and the duration of the attack decreased.

Second, measures to reduce the risk of operation: Fluids, preferably 5 to 10 per cent. glucose solution, are given by enteroclysis. The pre-operative administration of carbohydrates is probably the best insurance against post-operative hepatic complications. If the patient shows jaundice, special precautions are necessary which will be discussed under the head of biliary obstruction.

Pre-operative study in the doubtful or chronic cases includes an X-ray examination. Of the patients examined before the Graham technic was

adopted, 8 showed evidence of gall-bladder disease, while 18 gave no abdominal findings in a simple examination of the upper right abdomen.

Since using the Graham technic, a diagnosis of gall-bladder disease was verified in 12 cases, but in 3 others, cases in which a negative diagnosis was received, definite pathology was found in the gall-bladder region, an error of 20 per cent. In all of these cases the oral method of giving the dye was used.

Our opinion is, that an X-ray examination is not necessary in all cases of gall-bladder disease. It is more useful in the thinner patients who border somewhat on the ulcer type and in all cases in which the diagnosis is doubtful. The X-ray readings are correct in competent hands in about 80 per cent. of the cases. The patients the least likely to give a correct reading are the younger women in whom small stones have formed, but in whom the gall-bladder function is still good.

Operation.—1. *Anæsthetics.*—Operations on the biliary tract demand more care in the choice of anæsthesia than probably any group of abdominal operation. Gradually the tendency has been to change from the more shocking inhalation anæsthetics to the local and regional anæsthesia induced by novocaine, adding if necessary gas analgesia.

Anæsthetics

Ether—43 cases with 4 deaths.
Nitrous oxide and ether—48 cases with 3 deaths.
Local and gas—15 cases with 2 deaths.
Gas—1 case—no deaths.
Local—7 cases—no deaths.
Splanchnic—18 cases—no deaths.
Splanchnic and gas—4 cases—no deaths.

By far the most satisfactory anæsthetic in our experience is obtained by blocking the splanchnic nerves posteriorly as suggested by Kappis. There must be a local infiltration of the abdominal wall in addition, with a block of the intercostal nerves at the costal margin. After the peritoneum is opened a wall of local anæsthesia is placed just beneath the parietal peritoneum, using the finger in the abdominal cavity as a guide. It is usually necessary to place a small amount of novocaine in the round ligament and at times the anæsthesia is not complete until the gastro-hepatic omentum near to the pylorus and about the cystic duct is infiltrated.

The induction of this anæsthesia is somewhat time-consuming but is not painful or irritating except to the very nervous patient. It is successful alone in about 82 per cent. of the cases. In the others a light gas anæsthesia is all that is necessary. Under the splanchnic block it is possible to explore the abdomen, except the pelvis, and often the appendix can be pulled into the wound and removed without additional anæsthesia.

2. *Incision.*—The incision used in most of the earlier cases, and in all the cases in which the diagnosis was not certain, was one through the upper

right rectus muscle. There were 56 patients in whom this incision was used and they have all healed without evidence of hernia. The difficulty of exposure and of closure in many cases, and the frequency with which serum collected in the wound, led to a change.

In practically all of our later cases, a transverse incision was used extending from the rib margin nearly to the midline, at a level about two fingers' breadth above the umbilicus. After cutting through skin and subcutaneous fat, the obliques and transversalis are divided in the line of their fibres outward, beginning at the edge of the rectus sheath. The peritoneum is then opened and with the finger in the abdomen, two rows of interrupted sutures are placed, catching the rectus with its anterior and posterior sheaths. The rectus is then divided between the sutures to within three-quarters of an inch of its mesial edge. This incision usually gives sufficient exposure for cases of acute cholecystitis in which a cholecystostomy is to be performed. It may be readily enlarged by carrying the incision upward through the rectus. This is usually necessary in order to perform a cholecystectomy or an operation on the common duct.

The transverse incision was used thirty-two times and of this number there was one incisional hernia, and one patient who showed a slight pouting at the site of a cholecystostomy opening. There were 35 cases in which the incision was enlarged upward through the rectus, all of whom have healed without hernia.

This incision has the advantage of giving much better exposure than can be obtained by the right rectus incision alone. It is also very easy to close.

3. *Special Apparatus.*—In those cases in which cholecystostomy is to be performed, some instruments have been devised which greatly facilitate the operator, and which decrease considerably the operative trauma.

A sharp, round-pointed aspirating trocar, with water pump suction, devised by the junior author, is used to rapidly evacuate the fluid in the gall-bladder without soiling the wound, later a blunt-pointed aspirator may be substituted to completely empty the organ.

When the gall-bladder is empty of fluid and stones, the senior author's cholecystoscope may be used to view the inside of the organ. This instrument was used in fifteen cases. In that number there were four cases in which stones were discovered in the gall-bladder after a thorough search with scoop and finger had failed to show further calculi. An incomplete operation was discovered then in 26 per cent. of the cases in which the scope was used. We believe that from this group come most of the patients who have a return of symptoms following cholecystectomy.

Post-operative Treatment.—The patients are placed in semi-Fowler position and are given enteroclysis routinely, a pint each of tap water with tincture of digitalis two fluid drahms, glucose 5 per cent. and sodium bicarbonate 2.5 per cent. in order named. Since the use of local anæsthesia, fluid may frequently be given by mouth, after the first or second pint of enteroclysis.

Ill patients are encouraged to suck fruit lozenges or to use chewing gum, in an endeavor to keep the mouth moist and minimize the occurrence of parotitis.

Carbohydrate foods have proven the most helpful in these cases because of their rapid absorption and easy utilization by the body. Glucose with insulin intravenously we have found to be the best treatment for those cases of "liver deficiency" which appear a considerable time after operation.

The patient is instructed to take ten deep inspirations per hour. This has a tendency to clear the lungs and minimize pulmonary complications.

After the first week the cholecystostomy and common duct tubes are clamped for an hour after each meal, and the interval between clampings is decreased gradually so that toward the end of the second week the tubes may be clamped all the time. By thus allowing the bile to flow into the intestine during the periods of digestion, we have been able to overcome the nausea that frequently follows the taking of food in these cases of bile drainage.

In our cases the average time for the removal of the cholecystostomy tube was thirteen days. The choledochostomy tube, usually a T-tube, was removed from the sixteenth to the nineteenth day, *i.e.*, as soon as the tube could remain closed for several days at a time, without causing pain. No biliary fistulæ have resulted.

Hospital Days.—Cholecystostomy patients usually remain in bed until the tube has been removed (fourteen days). They are discharged after they have been up and around the ward for two or three days. The average stay in the hospital for these patients was twenty days, divided as follows:

Less than 15 days	16–19 days	20–29 days	30 days +
7 cases (1 in hospital)	13 cases	25 cases	4 cases

A cholecystectomy without drainage kept the patient in the hospital an average of seventeen days in 40 cases. These were divided as follows:

Less than 15 days	16–19 days	20–29 days	30 days +
23 cases (1 in hospital)	13 cases	4 cases	3 cases

A cholecystectomy with drainage demanded an average stay in the hospital of twenty-six days in 12 cases. These were divided as follows:

Less than 15 days	16–19 days	20–29 days	30 days +
2 cases	6 cases	4 cases	3 cases

More than half the cases of cholecystostomy demand a hospital stay of more than three weeks, while a cholecystectomy closed without drainage gives a patient a 50 per cent. chance of discharge in two weeks, and 85 per cent. of these cases can be discharged in two and one-half weeks. A cholecystectomy case with drainage demands a longer stay in the hospital, the delay in most cases being attributed to a wound infection.

Post-operative Complications.—Wound infections occurred eight times in this series, five times in a right rectus incision, once in a transverse, and twice in a right-angled incision.

Pulmonary Group.—Broncho-pneumonia occurred in 4 cases, and acute bronchitis in 2. These cases were all in the group with acute cholecystitis.

Lobar pneumonia—1 case with death.
Massive atelectasis—1 case with death.
Pulmonary embolism—1 case.
Empyema thoracis—1 case following pericystic abscess.

Cardio-vascular group:
Chronic myocarditis—1 case with death.
Femoral phlebitis—1 case.
Hæmiplegia—1 case.

Early hepatic insufficiency (liver shock, or toxic liver) as characterized by high temperature, pulse and respiration, low blood-pressure, cold moist skin, etc., occurred in six instances, one or two days following operation. Death followed in each case.

Late hepatic insufficiency occurred in 2 cases two to three weeks after operation. These cases are usually ones with common duct drainage and are characterized by lassitude, low blood-pressure, subnormal temperature, anorexia, and vomiting. One of these patients died.

Liver abscess—echmococcus—1 case.
Acute pancreatitis—2 cases with 1 death.

The most serious of these complications fall in the group of so-called hepatic insufficiency. Our knowledge of the cause of these conditions is meagre, hence we know very little concerning their treatment.

Analysis of the Cases with Follow-up Results.—1. Acute Cholecystitis.—This group includes all the acute and subacute cases of gall-bladder disease, as evidenced by elevation of temperature, leucocytosis, and acute tenderness and rigidity in the upper right abdominal quadrant. In 14 of the 28 cases a mass was palpable in the upper right quadrant.

The choice of operation in these cases depends largely upon the conditions found at operation. When the gall-bladder is tense and thickened, the gastro-hepatic omentum œdematous and the head of the pancreas indurated, by far the safer operation is a cholecystostomy. Cholecystectomy should be performed only where drainage of the gall-bladder can not be accomplished because of gangrene or for other reasons apparent at the time of operation.

Summary of the Follow-up Results for Acute Cholecystitis

Operation	No symptoms	Improved	Unimproved	Operative death
Cholecystectomy 10	9			1
Cholecystostomy 21	16	4		1
Cholecystostomy with drainage of pancreas 2	2			

Of the 4 patients listed as improved, one has had one attack of pain and vomiting of two days' duration, one indigestion, one has a slight hernia at the

site of the drainage tube, and one returned with a pericystic abscess followed by an empyema thoracis.

The mortality in this group was 6.06 per cent., with cholecystectomy 10 per cent., and with cholecystostomy 4.3 per cent.

Analysis of Cases that Died Following Operation for Acute Cholecystitis

Case	Age	Duration of acute symptoms	Operation	Post operative days	Cause of death	Necropsy findings
1	45	2 days	Cholecystectomy	1/6	Pulmonary embolism	Refused-obesity
2	65	20 days	Cholecystostomy Appendectomy	1	Acute hepatic insuf.	Hepatic cirrhosis. Chr. myocarditis. Chr. hypertrophic prostatitis. Extreme obesity.

2. *Chronic Gall-bladder Disease.*—This group comprised 78 cases. There were two cases in which an associated duodenal ulcer was found and for which a posterior gastro-jejunostomy was performed in addition to the removal of the gall-bladder. In 22 of these cases an appendectomy was also performed.

The choice of operation in this group is for a cholecystectomy, if it can be performed with safety to the patient. The operation should not be attempted if:

(1) A very fat abdominal wall prohibits good exposure.
(2) The patient is not a good operative risk because of jaundice, pancreatitis, cardiac or renal disease, age, respiratory infection, etc.
(3) The assisting and nursing staff are strange or inadequate.
(4) The anatomical difficulties found at operation are extraordinary.

In all these cases it is safer to drain the gall-bladder, *i.e.*, in case of doubt do a cholecystostomy.

Summary of the Follow-up Results for Chronic Cholecystitis

Operation	No symptoms	Improved	Unimproved	Operative mortality	No report	
Cholecystectomy 49	37	2	1	2	6	1*
Cholecystectomy Posterior gastro-enterostomy 2	1			1		
Cholecystostomy 27	18	6	1		1	1*
Cholecystotomy 2	2					

* In hospital.

Of the patients in which cholecystectomy was performed two patients were not entirely well. They complained of "bilious attacks," distention and belching after eating. They had no pain. One patient in whom the preoperative diagnosis was not clear complains of the same symptoms as before operation. She is a thin, neurotic individual and a regular attendant at the

medical dispensary. Of the 40 patients from whom follow-up reports have been obtained, 35 are entirely relieved—87.5 per cent. cures.

The operative mortality was 4 per cent. in cholecystectomy without associated disease. In the cholecystostomy cases without associated disease the mortality was nil, and the combined mortality 2.5 per cent.

There were 6 cases listed as improved following cholecystostomy. Of these two were free from pain, and complained only of belching and distention. Three patients had a return of pain, in one or more attacks, and a cholecystectomy was later performed on one of these. One patient has discharged several facetted stones from her drainage tract. There were no biliary fistulæ. The patient listed as unimproved later developed carcinoma of the gall-bladder.

Of the 25 patients followed, 18, or 72 per cent., are completely relieved by cholecystostomy. There were no deaths in the 26 cases.

This low morbidity figure is probably not quite a fair one in that many of these cases were operated upon but a short time ago (since September, 1922).

Analysis of the Mortality Following Operation for Chronic Gall-Bladder Disease

Case	Age	Duration of symptoms	Previous operation	Operation	Post-operative days	Cause of death	Associated conditions
1	41	3½ yrs.	Cholecystostomy	Cholecystectomy	1	Acute hepatic insufficiency	Mitral stenosis Incisional postneg.
2	50	1 yr.	None	Cholecystectomy and Appendectomy	2	Acute hepatic insufficiency	Incisional postneg.
3	40	2 yrs.	None	Cholecystectomy Posterior gastroenterostomy	4	Acute pancreatitis	Abdomen showed fat necrosis

Combining groups 1 and 2, the mortality with cholecystectomy was 5 per cent. in 58 cases, and with cholecystostomy alone it was 2 per cent. In other words, in the 111 cases of gall-bladder disease, both acute and chronic, without associated lesions, the operative mortality was 3.6 per cent.; adding the one death in the two cases with associated duodenal ulcer makes a mortality of 4.4 per cent.

3. *Chronic Gall-bladder Disease with Common Duct Obstruction.*—The patients in whom there is a biliary obstruction are among the poorest of surgical risks. Their pre-operative preparation should be as thorough and complete as is that practiced for patients with toxic goitre. It should include an estimate of bile pigments in the blood serum, urine and fæces in order to accurately follow the course of the jaundice after operation. The clotting time should be estimated and calcium chloride given intravenously as a routine measure. It is well to have a donor ready and cross agglutinated with the patient before operation.

The fluid intake should be kept high, giving hypodermoclysis if necessary.

Carbohydrate ingestion should be encouraged, by mouth, in solution, in the fluids given by enteroclysis or hypodermoclysis and even intravenously.

Splanchnic and infiltration anæsthesia is never more useful than in this group of cases. Ether seems to act poorly in these patients with damaged livers. Under splanchnic block a cholecystectomy and a choledochostomy may be performed, yet the temperature and pulse be within the normal range when the patient is returned to the ward.

Summary of Follow-up Results Following Operation for Chronic Cholecystitis with Biliary Obstruction

Operation	No symptoms	Operative mortality
Cholecystectomy with common duct drainage		
11	10	1
Cholecystostomy with common duct drainage		
8	5	3
Choledochostomy		
2	1	1

All of the patients who lived were completely relieved. The operative mortality was 23 per cent.

Analysis of Mortality Following Operation for Chronic Cholecystitis with Biliary Obstruction

Case	Age	Duration of symptoms	Duration of jaundice	Operation	Position of stones	Post-operative days	Cause of death	Associated conditions
1	44	10 yrs.	Slight Intermittent	C. ostomy C. dochostomy Appendectomy	Common duct above duodenum	5	Lobar pneumonia	Increase of jaundice Obesity
2	37	5 yrs.	7 wks.	C. ectomy C. dochostomy	Common duct above duodenum	6	Massive atelectasis	None Obesity
3	54	1 yr.	7 wks.	C. ostomy C. dochostomy	None Stricture at junction of cystic and hepatic duct	14	Hepatic insufficiency	Obesity
4	27	9 yrs.	2 wks.	C. ostomy C. dochostomy	Behind duodenum	1	Acute hepatic insufficiency	Necropsy showed hepatic cirrhosis
5	50	7 yrs.	1 wk.	C. dochostomy	Behind duodenum	1	Acute hepatic insufficiency	Necropsy showed hepatic cirrhosis

There is little choice in the operation in these cases. The common duct is always opened and the obstruction relieved by the removal of the stones. To

make sure that the duct is patulous a small French catheter is usually passed through the sphincter into the duodenum. The common duct is usually drained by the use of a T-tube. If conditions allow, the gall-bladder is emptied of stones and a tube sutured in it. In these jaundiced cases the gall-bladder is not removed if it can be used for drainage. In some cases the gall-bladder shows so much damage that it must be removed and in other cases it may be so atrophic that it can be allowed to remain untouched. The least that can be done to relieve the immediate emergency in these cases offers the best chance for recovery.

4. *Carcinoma of the Gall-bladder.*—There were two cases in this group, an incidence of 1.4 per cent. for the 136 patients. Both of the patients were males, one fifty-four and the other sixty-one years of age. A history was obtained of upper right-sided pain of rather short duration and a mass could be palpated in the right hypochondrium in each case.

In one patient a cholecystostomy was performed without recognizing the carcinoma. Enlarged glands were noted about the cystic duct which were thought to be inflammatory. He returned one month after his discharge with complete biliary obstruction. At operation there was a mass of indurated tissue in the region of the gastro-hepatic omentum, but no evidence of common bile duct could be found. The patient died in the hospital fourteen days after operation.

The second patient was diagnosed as carcinoma of the gall-bladder before operation. A cholecystostomy was performed for the relief of pain. No attempt could be made to remove the gall-bladder because the growth had already invaded the liver. The patient died at home one month after operation.

SUMMARY

1. An analysis has been made of 136 cases of gall-bladder disease of whom 94 + per cent. have been followed.

2. A theory has been advanced against the infectious origin of cholelithiasis.

3. The surgical principles used in the treatment of these cases have been enumerated.

4. Of the cases of acute cholecystitis, 89 per cent. have been cured by cholecystectomy with an operative mortality of 10 per cent.; by cholecystostomy, 76.4 per cent. have been relieved of all symptoms, with an operative mortality of 4.3 per cent. Combined—6 per cent.

5. Of the cases of chronic cholecystitis, 87.5 per cent. of the patients who had a cholecystectomy were cured with an operative mortality of 4.1 per cent.; cholecystostomy resulted in no mortality and 72 per cent. of the patients were relieved. Combined 2.5 per cent.

6. The operative mortality of the combined acute and chronic infections of the gall-bladder unassociated with other lesions was 3.6 per cent.

7. Of the patients operated on for biliary obstruction 77 per cent. were relieved. The operative mortality was 23 per cent.

FOREIGN BODIES IN THE INTESTINE*

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Operation for an acute perforation of the sigmoid, produced by a pin accidentally swallowed six days previously, disclosed the fact that the head and not the point presented itself through the wall of the gut. The literature of foreign bodies in the intestine is replete with interesting and unusual cases such as this. Ingested foreign bodies, varying widely in number, size, and character, may travel through a distensible tube of comparatively small diameter, causing repeated mechanical insults to the intestinal wall without producing symptoms or permanent tissue damage. In addition, the phenomena mentioned above will be considered from the standpoint of the pathological physiology and the mechanical factors involved.

From 1915 to 1926, there have been at the Presbyterian Hospital forty-eight cases of proven foreign body in the intestine. All of them were swallowed and many different types were encountered. Most of those that could be followed were evacuated without untoward symptoms. For purposes of simplification the cases have been charted according to a definite scheme which is seen in the accompanying table.

An analysis of these cases (also seen in the graphs, Figs. 1 and 2) shows the following:

ANALYSIS OF 48 CASES †

Age:

Youngest patient	5 months
Oldest patient	64 years
Number of patients between:	
1–5	31 = 64.7 per cent.
6–10	5 = 10.3 per cent.
11–20	4 = 8.3 per cent.
21–65	8 = 16.7 per cent.

Sex:

Female	26 = 54.2 per cent.
Male	22 = 45.8 per cent.

Type of Foreign Body:

Sharp	Pins	Straight pin	6	False molar with plate	2
		Bar pin	2	Tack	1
		Pin with large head	3	Nail	1
		Screws	4	Fishbone	1
		Needle	2	Chicken-bone	1

Total .. 23 = 47.92 per cent.

* Read before the Section on Surgery of the New York Academy of Medicine, December 3, 1926.

† The common factors for all these cases were age, sex, and type of foreign body. Thirty-one cases could be followed completely and these are analysed separately with additional constant factors.

Dull
- Coins: Penny ..... 5; Nickel 3
- Closed safety pins 4
- Toys: Whistle ..... 2; Metal horse 2
- Fruit stone, seeds 3
- Ring 2
- Pencil 1
- Piece of metal 1
- Stomach tube 1
- Tube with radium 1

Total ..... 25 = 52.08 per cent.

ANALYSIS OF 31 CASES (WITH FOLLOW-UP)

Number of sharp foreign bodies that passed without complications—10 out of 12 = 83.3 per cent.

Number of dull foreign bodies that passed without complications—15 out of 19 = 79 per cent.

Total number passed—25 out of 31 = 80 per cent.

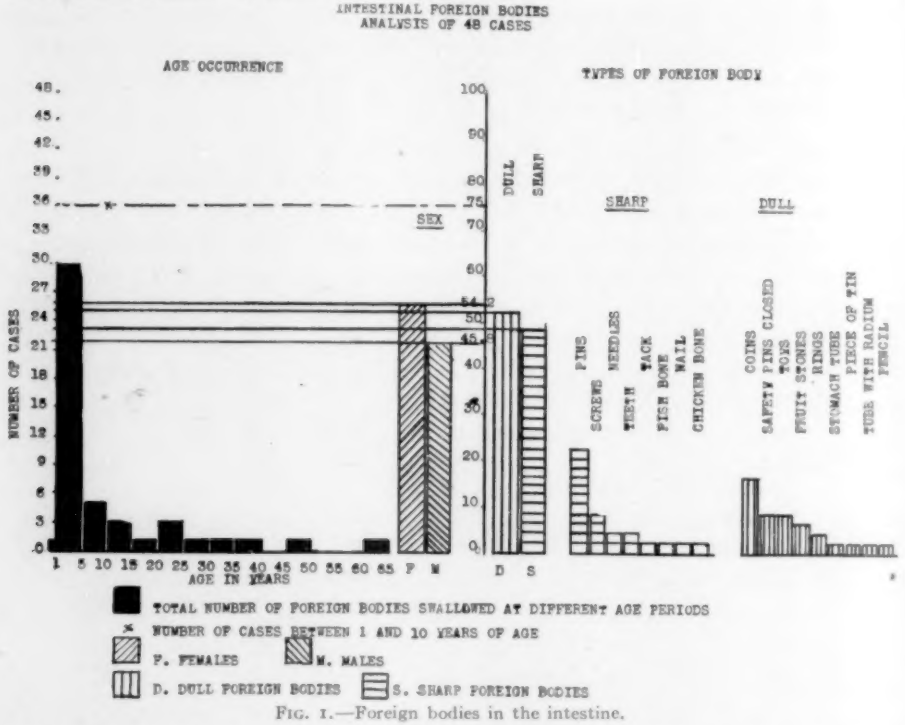

FIG. 1.—Foreign bodies in the intestine.

Symptoms referable to those foreign bodies that were passed—4 (16 per cent.) had symptoms divided between abdominal pain and vomiting.

Symptoms referable to complications:

1. Acute ileus—case died.
2. Chronic ileus—case died.
3. Perforation of sigmoid with peritonitis—case recovered.
4. Acute appendicitis—case recovered.
5. Pelvic peritoneal abscess—case recovered.

Longest time that foreign body stayed in any one part of intestine.

Nail in cæcum—3 weeks.

Shortest time in which foreign body was passed: dull—20 hours; sharp—2 days.

Longest time in which foreign body was passed: dull—4 weeks; sharp—3 weeks.

Average time for passage of sharp foreign bodies—7.33 days.
Average time for passage of dull foreign bodies—5 days.
Average time for passage of all foreign bodies—6.17 days.
35 per cent. of all the cases had catharsis. A little less than half of these had sharp foreign bodies. None developed complications.
To summarize, it appears from the foregoing analysis that:

1. The foreign bodies were swallowed accidentally or by those who knew no better.

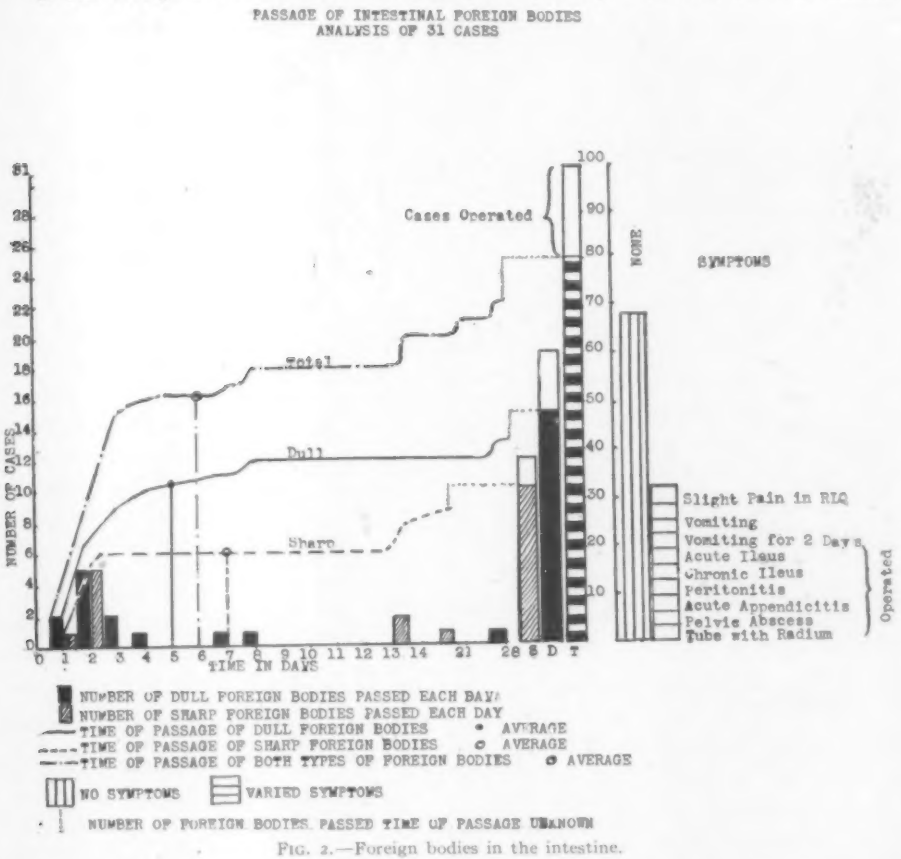

FIG. 2.—Foreign bodies in the intestine.

2. The largest number (75 per cent.) occurred among babies and children under ten years of age.
3. The cases were about equally divided between the sexes (not very significant in view of No. 2).
4. Dull objects were slightly preponderant.
5. Among sharp objects pins were preponderant.
6. Most of the foreign bodies (80 per cent.) were passed.
7. Of the sharp foreign bodies only two (15 per cent.) perforated the gut.
8. The minority of cases that pass foreign bodies have symptoms or signs.
9. It takes a sharp foreign body a little longer to pass than a dull foreign body.

Practical Anatomy and Movements of the Intestine.—The intestinal canal is a long, elastic, distensible, and motile tube, varying in diameter at different portions and characterized by natural folds in its wall, valvular formations, angulations, and mobility throughout its greater extent. Nor-

mally, it offers points of possible delay or obstruction to a foreign body as follows:

1. The junction of the second and third part of the duodenum, due to the flexure and the sphincter muscle described by Ochsner.

2. The ileocæcal region because of the angular insertion of the ileum.

3. The lumen of the appendix, which may harbor a foreign body due to incompetency of the ileocæcal valve.

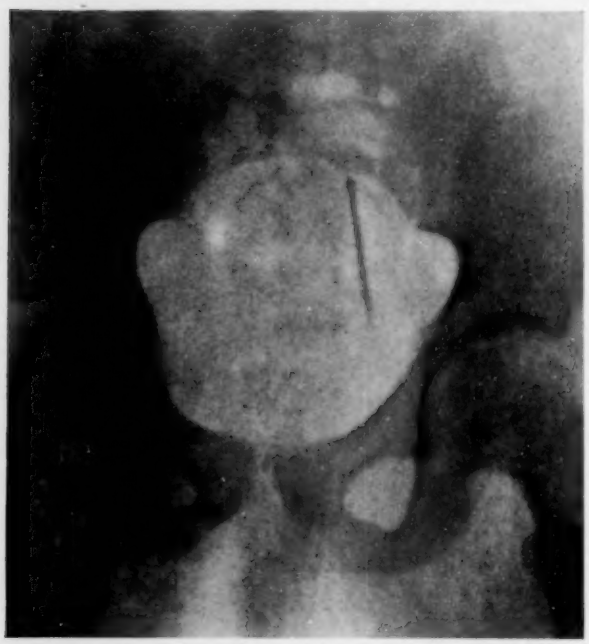

FIG. 3.—X-ray picture of pin in intestine of eleven-year-old girl the head of which perforated the sigmoid six days after it was accidentally swallowed.

4. The junction of cæcum and ascending colon, because of the presence of the "frenulum valvulæ coli."

5. Flexures and haustræ of the large intestine, including the rectal ampulla and crypts of Morgagni.

In order of frequency, the rectum, cæcum and sigmoid probably offer the best anatomical sites for the arrest of a foreign body. The movements of the intestine which normally are controlled centrally, through extrinsic nerves, and peripherally, by the neuromuscular mechanism in the wall of the intestine, may be grouped under the following heads:

1. Long tonic contractions (pendular movements).
2. True peristaltic contractions.
3. Rhythmic segmental contractions.
4. Antiperistaltic contractions, present mostly in the large intestine.

A foreign body might normally encounter a bar to further progress by one of the anatomical factors mentioned above. On the other hand, the tendency is for propulsion forward by intestinal movements, which in themselves might cause the foreign body to come in intimate contact with the intestinal wall. Nature's usual protection and reaction against this contact will be discussed later.

Reference to Poulet's [1] classic on foreign bodies in general and to Peter's [2] short monograph will give many amazing, unusual, and curious examples of foreign bodies in the intestine. Their presence may be due to migration from a neighboring organ, cavity or extremity, to accidental or deliberate ingestion or to introduction through the anus into the rectum. Deliberate

ingestion may result from an act of insanity, a dare, a habit or medicinal therapy. Accidental swallowing of foreign bodies is by far the most common. Fish bones, chicken bones, and fruit pits, normally present in food, are frequently swallowed in careless and rapid eating. On the other hand, individuals may unknowingly swallow foreign bodies such as slivers of wood in bread. A foreign body placed in the mouth temporarily may be swallowed through absentmindedness or a sudden inspiratory effort. I recall a patient who went to sleep with a toothpick in his mouth and swallowed it. In our series, St. John removed from the duodenum 50 mgs. of radium in a brass container. This had been placed in the nose for an ethmoidal condition and was accidentally swallowed.

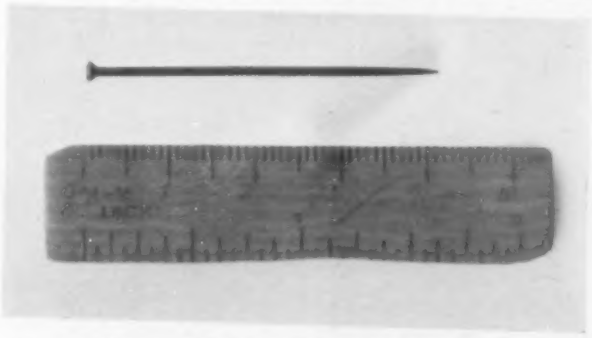

FIG. 4.—Pin shown in Fig. 1 after operation.

The ingestion of foreign bodies by the insane is a common occurrence. Ross [3] reports a case of an insane woman, who, after a six months' history of lower abdominal pain was found to contain twenty pieces of wire in a resected piece of ileum. Stough [4] cites a case of an adult insane male who swallowed a knife six and one-quarter inches long with a two and one-quarter inch blade. In several months a fecal fistula formed in which the knife was found. A female inmate of sixty-six was autopsied by Hill.[5] A palpable mass in the right iliac fossa, the size of a tangerine orange, was found to be a hair-ball in the ileum. Hosford [6] reports post-mortem findings on a lunatic, who, while alive, had been known to pass stones and pieces of wood and cloth. Autopsy showed innumerable articles, including wood, linens, neckties, and stones, in the jejunum, ileum and sigmoid, with a marked chronic pelvic peritonitis. It is remarkable that there was no ulceration or perforations of the gut wall. During a laparotomy on a neurotic woman, presumably for appendicitis, Gray [7] found the bowel perforated by a hairpin which had entered the abdominal wall. There were present also ten and one-half hairpins, seventy-eight ordinary pins, one nail, and one piece of steel three-quarters of an inch long. The patient finally admitted that she was in the habit of putting one or two pins in her mouth before going to bed in the event that she should awake and require them.

Pringle [8] records the case of a man of twenty who swallowed seven nails one inch long "on a dare." Eight days later he came to operation and the nails were found in the cæcum. He died of a post-operative ileus and autopsy showed early necrosis of cæcum and ascending colon. Another patient (Genglaire [9]) swallowed thirty frogs and had no symptoms until they reached the rectum. A large mass of tangled frog bones was extricated manually.

Material taken for therapeutic purposes such as bismuth or bran might accumulate in an already pathological colon and eventually create a foreign body. Vegetable fibres have been known to do the same. It is a well-known fact that the residuum of oatmeal, consumed in large quantities by many inhabitants of Ireland, may cause intestinal obstruction.

Women and babies occasionally make a habit of swallowing one particular

type of foreign body. As an illustration, we saw a colored child who was accustomed to pluck its own crimpy hair and swallow it. In 1914, Heazlit [10] reported 70 cases of hair-ball in the gastro-intestinal tract which he had collected from the literature. The habit of hair swallowing is practically confined to females, who are, as a rule, normal mentally. Operative therapy is usually necessary to relieve symptoms.

We will mention only in passing, the presence of gall-stones or scybalous masses in the intestine.

Although almost any type of foreign body may gain access to the intestine, those most frequently found are metal, bone, fruit pits, glass, hair, wood and cloth. Deliberate ingestion, usually accomplished with less choking and pain than accidental ingestion, accounts for the remarkable size of some of these bodies. A 6½-inch dinner fork with a 4-inch handle was swallowed by a female of twenty-five,[11] a 2½-inch scarf pin with a small glass ball at one end by a child of two,[12] a closed pocket knife 2½ inches long by a child of seven months,[13] a 2-inch iron nail by a two-year-old baby,[14] a 3¾-inch toothbrush by an infant eight weeks old,[15] and in our series a 3-inch nail with a large head by a boy of six.

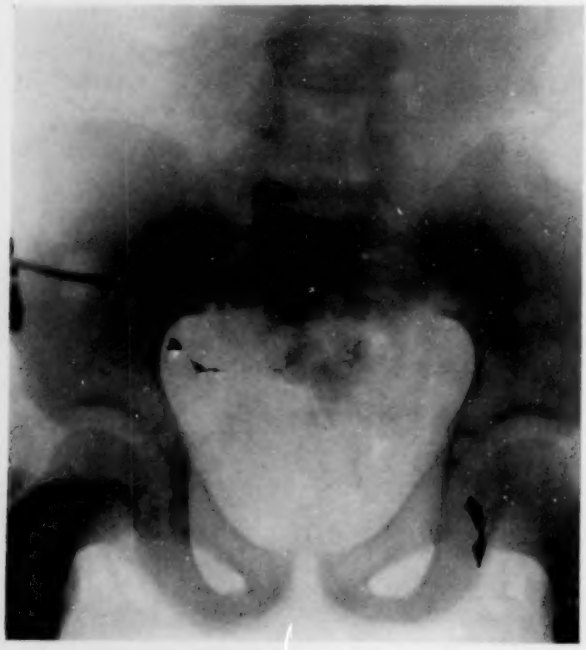

FIG. 5.—Nail which remained in cæcum of six-year-old boy for three weeks and was then suddenly passed after a barium enema.

What happens to the foreign body in the intestinal lumen and the reaction that may be produced in the intestinal wall constitute a most important aspect of this entire subject. Many are recovered in the same condition as before ingestion, but the intestinal juices may cause a metal object to break in two; or foreign bodies may be surrounded by some natural protective coat consisting of mucus, unabsorbed food or fæces. Glass is usually rounded off by the digestive juices, and Simmons and Von Glahn [16] found that the ingestion of ground glass has no toxic effect nor does it produce any permanent lesion on the gastro-intestinal tract of dogs. Faber [17] gave food with fishbones to adults, some of whom had a normal gastric acidity or hyperacidity and some of whom had a subnormal acid gastric content. In the former group the fæces showed no fishbones, while they were present in the latter. He infers that in the first instance normal decalcification could take place.

Although nature's protection facilitates the passage of most foreign bodies, symptoms frequently develop from obstruction, traumatism to or perforation of an organ. Thus we may find a peritonitis, peritoneal abscess, or fistulous communication between intestine and intestine, or between intestine and some other organ, such as the bladder. Other foreign bodies that are thin and sharp may perforate the gut, producing few or no symptoms, and travel through the peritoneal cavity or along muscle planes or into a large blood-vessel. Such a procedure usually takes a long time. David [18] cites

FIG. 6.—Unknown type of foreign body causing intestinal obstruction.

a case of an adult who, after swallowing a fishbone, had a cystitis for twenty-nine months. The bone was then passed per urethram, having migrated from the rectum into the bladder. Cordier [19] reports the case of a boy of sixteen, who had had an attack of pain in the right lower quadrant three years previously, in whom a diagnosis of typhoid fever was made. Subsequently he had urinary tenesmus with tenderness in the hypogastrium. Two stones were found in the bladder, one of which was incorporated with a veil-pin which had protruded into the bladder. A sane but neurotic male of thirty was operated on by Bell [20] for abdominal pain. He found a needle with one end in the stomach and the other in the liver, another needle free in the gastrocolic omentum, and a 4-inch hatpin piercing the duodenum. Nine years after a man had swallowed a darning needle, it was extracted from the posterior aspect of the hip-joint (Woodman,[21]). Mueller [22] had a case in which a pin was found lying across the ureter nine years after ingestion. In a woman who came to autopsy after having had an œdema of both lower

extremities, Thompson [23] found a needle with a surrounding thrombus in the inferior vena cava. Many cases have been operated presumably for appendicitis, in which a foreign body in or near the appendix was found to be the real etiological factor, Pike,[24] McCrae,[25] Speese,[26] Hertz.[27] In an analysis of 63 cases of foreign body appendicitis, Fowler [28] states that, of the larger foreign bodies, straight pins are found most frequently and that the symptoms are chronic in the majority of cases. Bidwell [29] found foreign bodies in about 20 per cent. of the appendices that he removed, and he believes that symptoms were produced not so much by the foreign bodies as by concretions formed around them.

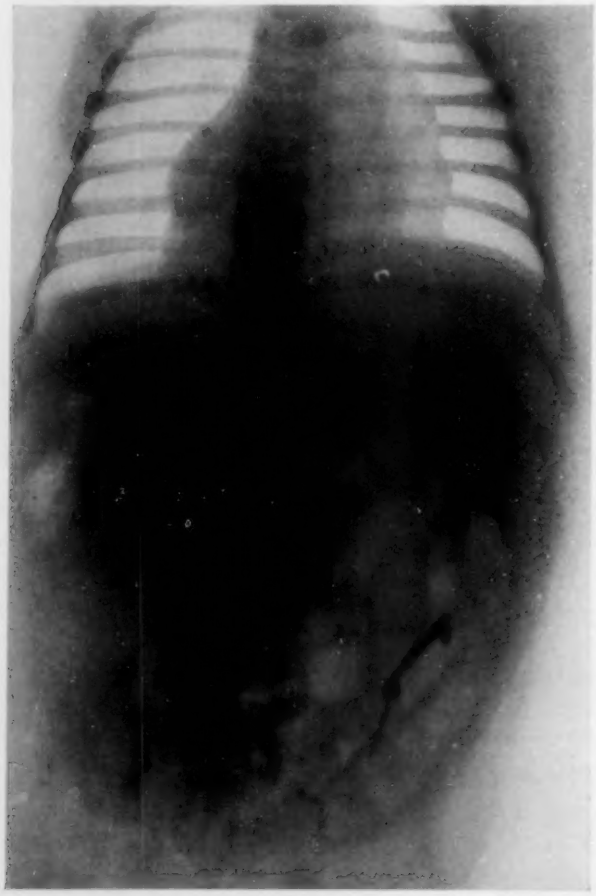

FIG. 7.—Röntgenogram of open bar-pin in intestine of five months old baby, passed in three days.

Two Italian observers, Zoja [30] and also Omboni,[31] were impressed with the usual favorable outcome of foreign bodies in the intestine in humans and experimentally in dogs. To ascertain the reaction of the intestinal wall to foreign bodies which permits their successful progress and evacuation with such remarkable frequency, Exner [32] conducted a series of beautiful and carefully controlled experiments on the intestine of dogs and cats. He found that if the mucosa of the small intestine is stroked or pricked, an anæmic area is formed after a period of time varying from several seconds to two minutes. In the centre of this area there is an inconstant concavity surrounded by a muscular and contractile wall. Reference to Fig. 10 will show the reactions noted in confirmatory experiments on the dog's intestine. The same results were noted when the experiment was performed with the sero-muscular layer removed. In both cases these phenomena disappeared in about one minute. In the second experiment the histological examination showed that the concavity was due to contractions

of the muscularis mucosa and of the fibres between the lymph follicles and the glands, and that the anæmic area was produced by compression of the blood-vessels caused by these contractions. An exsected piece of intestine showed the same reactions as above, proving that the ganglia in the intestine itself are the controlling factors in their production. These results seem to be less constant in the large intestine.

Continuing his experiments, Exner proceeded to feed long-pointed glass splinters to cats and found that the places where the glass touched the stomach wall were drier than the surrounding mucosa. There was no wound, but at the point of contact a concavity was formed in an area of anæmia. In the small gut the glass was also embedded in concavities, but in the large intestine they were found in the natural folds. The serosa presented nothing abnormal.

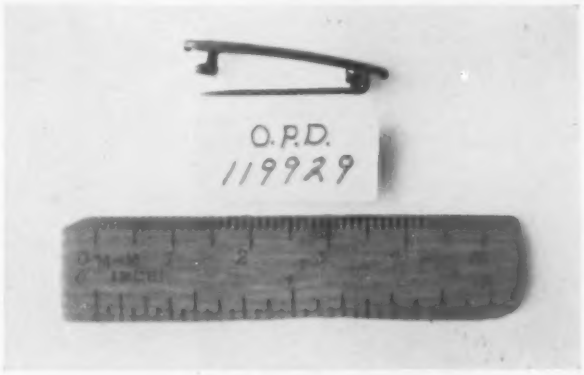

FIG. 8.—Pin seen in Fig. 7.

A dog on which he experimented was given food in which fifty needles were incorporated, one-half of which had their points isoperistaltic and the other half points antiperistaltic. After twenty-four hours, forty-eight needles had been passed with the heads isoperistaltic and the other two with the points isoperistaltic.

Summarizing other experiments, about 800 needles were introduced, points isoperistaltic, into the stomachs of dogs and cats. All the animals were well before they were killed, and autopsy showed no peritonitis, and no visible injury to the mucosa. Furthermore, the number of needles passed with heads isoperistaltic were found in a ratio of 7 to 3 that were passed with points isoperistaltic. This means that the intestine has a tendency to pass pointed foreign bodies with blunt end forward.

The significance of these observations brings us to the following consideration of the reasons why pointed foreign bodies in the majority of cases proceed through the intestinal tract with heads isoperistaltic and are passed through the anus in the same position:

1. If the contents of the intestine are entirely fluid, the centre of the column moves faster than the periphery. At the mucosa, where the point of a foreign body may impinge, the rate of movement of the fluid content is practically negligible, and therefore the head, which is in the faster current, is pushed forward.

2. If the contents of the intestine are firm, and if a pin, traveling with the point forward, touches the mucosa, a concavity forms between the mucosa

and column of fæces at the point of contact, and the fecal mass pushes the head of the pin in an isoperistaltic direction.

3. The muscular boundary of a concavity may stop the point of a pin and the adjacent fæces, causing part of the fecal mass to turn and force the head forward.

4. The point of a pin may get caught in the natural folds of the gut, and any of the three preceding factors may operate to push the head of the pin forward.

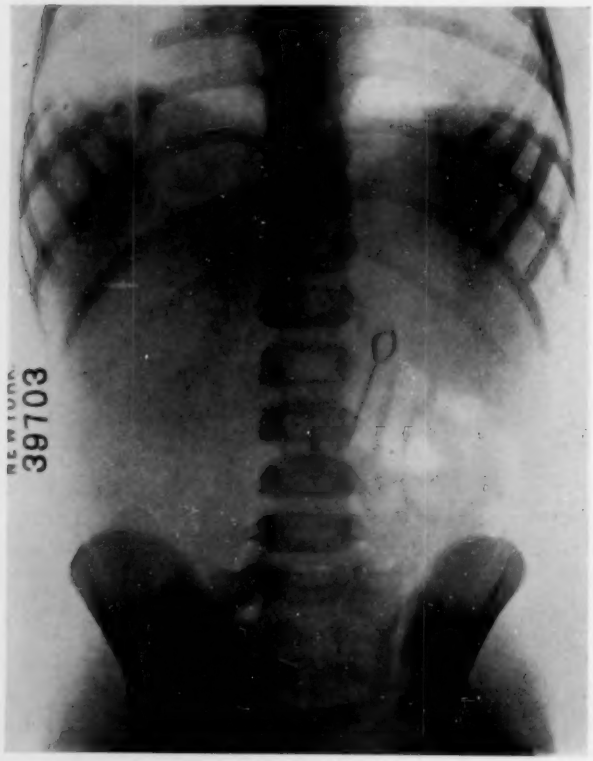

FIG. 9.—Pin in intestine of three-year-old child, passed in fourteen days.

With this knowledge of the mechanism of nature's protection against injury or perforation of the intestinal wall by foreign bodies, it is easy to understand how some foreign bodies may lodge in the distensible tube for a long time, especially when they have been completely enveloped by undigested food particles or fecal material. In a six-year-old boy in our series a 3-inch nail apparently stayed in the cæcum for three weeks and then was suddenly passed. Smith [33] reports a case of a woman who swallowed a darning needle 1 11/16 inches long and passed it twenty years later!

In about 90 per cent. of the cases of sharp foreign bodies analyzed in our series there was no perforation. When perforation does occur it probably is caused by pressure necrosis superinduced by a local inflammatory process, especially in the large intestine, where bacterial flora are profuse. Violent peristalsis or the act of defecation might drive the foreign body into the intestinal wall. Anatomical and pathological conditions, such as the narrow lumen of the appendix, the sacculations of the large gut, carcinoma, tuberculosis, diverticula, hernia, or any factor causing a chronic obstruction, will clearly predispose to perforation. Perforation in the large intestine is more frequent since the narrower lumen of the small intestine makes it difficult for the foreign body to turn around and its slow vermicular action makes lodgement or perforation here unlikely.

Most foreign bodies in passing through the intestine produce no symptoms. When symptoms occur the diagnosis must be made on a careful history and a physical examination followed by the various laboratory aids, especially the X-ray. Such symptoms may be summarized as follows:

1. A mild cramp when they pass through the intestine naturally.
2. A cramp or pain in one spot when they pass through slowly.
3. Diarrhœa and mucus or blood in the stool from irritation of the intestinal mucosa.
4. Intestinal obstruction.
5. Pain, tenderness and constitutional symptoms from inflammation and pressure necrosis.
6. Perforation.

Treatment.—The immediate course to be pursued following the ingestion

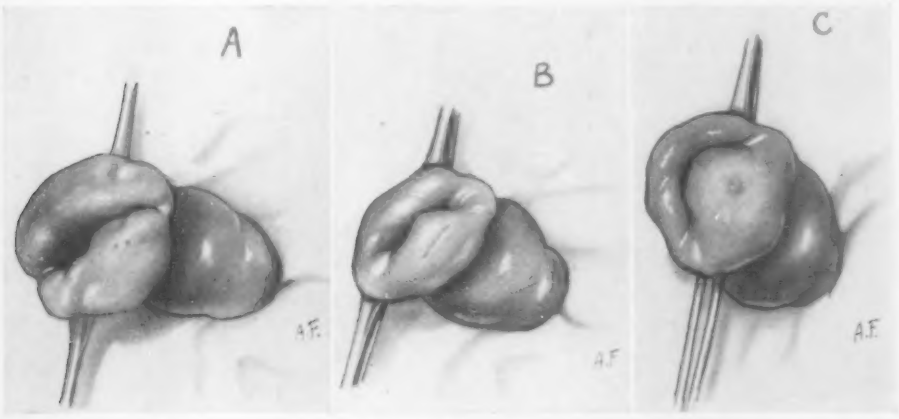

FIG. 10.—A. Showing two small concavities produced on the mucosa of small intestine of dog by gentle pricks of needle point. They were preceded by temporary area of anemia and appeared in about two minutes, gradually becoming deeper and slowly disappearing in about ten minutes. B. Similar trough-like reaction produced by gentle stroke of needle point. It disappeared in about fifteen minutes. C. Concavity with surrounding area of marked anemia produced by pushing into mucosa gently with blunt end of artery clamp. The anemia persisted for about twenty minutes, and the concavity fifteen minutes.

of a foreign body is expectant. The size and the nature of the foreign body, the possibility of its localization, and the condition of the intestine itself will decide the wisdom of a subsequent radical procedure. The fear and the actual danger of the potential harm from the foreign body are never to be discounted. The unexpected may occur at any time from an apparently innocent foreign body. Close observation and immediate operative therapy when the symptoms so warrant are of paramount importance. On the other hand, we have shown by statistics and by experimental work that there is a natural tendency for the spontaneous passage of foreign bodies without untoward symptoms. To help nature, two factors are essential: the prevention of intestinal hypermotility, and the ingestion of such material as might aid in the formation of a protective coat around the foreign body. The idea of hastening the exit of a foreign body by the use of a cathartic is, I think, a mistake. Powerful intestinal contractions may drive the foreign body into or through the intestinal wall. Bran, agar-agar, whisps of cotton, pultaceous

Synoptical Table of Cases of Foreign Body in the Intestine

Hospital No.	Age	Sex	Foreign body	Symptoms	Treatment	X-ray findings and fluoroscope	Result	Complications
67277	9	Male	Melon seed and hairs in appendix	Acute appendicitis	Appendectomy		Improved — perhaps foreign body cause of symptoms. As examination of appendix and abdomen negative	
Dr. Carp Private case	11	Female	Bank pin	None until 7th day—acute lower abdominal pain with signs of peritonitis	Dietetic until perforation, no castor oil—cereals, agar-agar, bread	Six days after ingestion pin in L. L. Q.	Cured after operation (laparotomy, extraction of pin drainage)	Head of pin perforated sigmoid, local peritonitis.
50788	64	Female	Numerous foreign bodies (fruit seeds, leaves, fecalites)	Previous acute ileus (volvulus of small intestine) followed by operation with reduction of volvulus. Chronic ileus followed by an acute attack	Ileostomy (13 days later) Removal of foreign bodies. Removal of specimen		Died from post-operative complications	Chronic ileus and ulcer of colon.
55115	35	Female	Prune pit covered with calcium phosphate crystals	Acute ileus	Exploratory operation		Died	(Autopsy) Foreign body impacted in sigmoid at point of stenosis by adhesions—resulting ulcer—and perforation—associated acute diphtheritic colitis.
62323	24	Male	Piece of bone (bird?) 2.5 cm. long and 2 mm. in diameter	1. Pain in L. U. Q. radiating to L. L. Q. and lasting 3 weeks 2. Palpable mass in L. L. Q.	Incision and drainage of pelvic abscess with removal of foreign body. (Abscess surrounded by large coils of small intestine and sigmoid)		Improved	Peritoneal abscess
64370	15	Female	50 mg. radium in brass container which screened all but gamma rays. (Radium had been placed in nose for an ethmoidal condition)	None	Enterostomy, enterorrhaphy, and removal of foreign body from duodenum (operated on same day as swallowed)	X-ray—radium tube in region of stomach (apparently)	Cured	

51797	6	Male	Nail—3" long	Slight pain in R. L. Q. —of short duration	Castor oil by mother. Bowels kept open by enemas when needed. Given barium enema day before passing of foreign body	Two days later in cæcum. Repeated X-rays during following 3 weeks, nail apparently remaining in cæcum	Passed 3 weeks later	
23350	3	Female	Pin with large top	None	Mucilagenous foods and cotton	In second portion of duodenum	Passed 14 days after ingestion	
17807	11 mos.	Female	Safety pin (closed)	Vomited once, otherwise negative	None mentioned	Pain in abdomen	Passed 3 days after ingestion	
30458	2½	Male	Screw ¾" long	None except for slight tenderness in R.L.Q.	Soft diet containing much residue plus mild cathartic	Screw in pelvis 24 hours later	Passed screw 3 days after ingestion	
94811	1 yr. 8 mos.	Male	Penny	None	Castor oil	Fluoroscope ½ hour later—penny in fundus of stomach	Passed in 20 hours	
130507	2	Male	Toy metal horse ½" square	None	Castoria	Foreign body in duodenum ½ hour later. In lower right quadrant (cæcum or terminal ileum one day later	Passed 2 days later	
84898	16 mos.	Female	Lead pencil 3 cm. long, blunt point	None	Dry bread, prune juice, S.S. enema		Passed in 24 hours	
18274	3	Female	Tin not painted, 1" x 1½"	None	Castor oil (by mother) vegetable diet—agar-agar	24 hours later, foreign body seen in R.L.Q.	Passed in 74 hours	
120086	2	Male	Tack	None	Rest, bulky food, mild cathartic	10 minutes later, tack in stomach	Passed in 3 days	
80997	2	Male	Penny	None	Castor oil		Penny appeared in stool 2 days later	
119929	5 mos.	Male	Bar pin (open) 1¼" long	None		On same day pin seen in stomach. Pin in R.L.Q. 2 following days	Passed 3 days after ingestion	
136220	5	Female	Straight pin	None	Bed, no cathartic	Pin in decending colon or proximal sigmoid 2 days later	Passed pin 3 days later	
63672	16 mos.	Female	Screw	None	Bread and castor oil	Foreign body in lower abdomen one day after ingestion	Passed foreign body 2 days after ingestion	
79560	7	Female	Penny	Vomiting 2 days	Castor oil		Passed in one week	
86626	5	Female	Safety pin (closed)	None	Mashed potatoes, pan cakes, water	Pin in small intestine to left side of abdomen below umbilicus—6 hours after ingestion	Pin passed 2 days after ingestion	

SYNOPTICAL TABLE OF CASES OF FOREIGN BODY IN THE INTESTINE—*Continued*

Hospital No.	Age	Sex	Foreign body	Symptoms	Treatment	X-ray findings and fluoroscope	Result	Complications
147269	2½ yrs.	Female	Safety pin (closed)	None		Pin in L. U. Q. (fundus of stomach) 2 days after ingestion. 4 days later pin in same position	Passed 8 days later	
138862	14 mos.	Male	Safety pin (closed)	None		Pin in stomach same day	Passed 2 days later	
136220	5	Female	Straight pin		Bed, no cathartics	Pin in stomach with head toward pylorus, ½ hour later. Pin in descending colon on proximal sigmoid 2 days later	Passed 3 days later	
130507	2	Male	Toy horse (metal) ½" square	None	1 tsp. castoria	In duodenum, 15 minutes later. In cæcum or terminal ileum 1 day later	Passed 2 days later	
129435	3	Male	Penny	None	Castor oil	None	Passed in 4 days	
142431	24	Female	Needle	Pain in R. L. Q.	Cathartic	Needle in L. U. Q.	Passed in 2 weeks	
128735	3½	Male	Nickel	Pain in R. L. Q.	Cathartic	Needle in stomach	Passed in 4 weeks	
55412	27	Male	Intra-nasal feeding tube	None	Fluids by mouth. *Exploratory operation 14 days later*—tube found in cæcum but not removed	*4 days* later—object in duodenum, *23 days* later—in terminal ileum or rectum, *28 days* later—no evidence of tube	Tube is discharged—date unknown	Infection of abdominal wound healed 24 days later.
118098	16	Male	Straight pin	None	Bulky food	Fluoroscope—one hour later shows pin in stomach	Passed—time unknown	
7914	5	Female	Whistle	None	Large meal followed by emetic (advice of Dr. Lambert who believes foreign body will not pass pylorus)	3 days later foreign body in stomach	Passed in several days	
68435	3½	Male	Ring	None	Food with much carbohydrate	Ring in stomach or duodenum 4 days after ingestion	Lost to follow-up	
19844	10 mos.	Female	Pin with large top	None	Soft diet with wisps of cotton in oatmeal—also potatoes	Two days later pin in stomach	Observed in ward for 6 days—foreign body not noticed in stools	
48281	8½	Female	Hair pin 3" long	Nausea and pain in epigastrium for 2 days	Castor oil		Lost to follow-up	

98275	26	Female	Tooth with sharp screw attached	Vague pains in epigastrium	Diet		Lost to follow-up	
96197	2½	Male	Nickle	None	Castor oil (by mother)—bulky food	Following day foreign body in L.U.Q. In R.L.Q. (cæcum?) 1 week later	Not passed after 1 week's observation	
54873	38	Female	Fish-bone	None—symptoms of existing ulcer and gall-stones	Gastro-enterostomy for ulcer (duodenal) and gall-stones—fish-bone found in course of operation, situated subserously and no ulcer in its vicinity	None pertaining to fish-bone	Improved	
55871	14	Female	Pin	Some generalized abdominal tenderness		Pin left in left iliac fossa at junction of descending colon and sigmoid—few minutes later shadow seen 3" higher in descending colon	Lost to follow-up	
			NOTE.—No symptoms, treatment, etc.—foreign body discovered in course of general physical examination in a case of acute rheumatic fever.					
18222	2 yrs. and 9 mos.	Female	Needle	None at first, later constipation and one vomiting	Regular diet with oatmeal and potato	Two days later needle in L.U.Q. Repeated X-rays showed needle in same locality	Discharged as unimproved—case not followed	
7520	25	Male	False molar tooth with plate attached (2 cm. long)	Immediate "sticking" sensation in left sub-diaphragmatic region. Otherwise negative	Bulky food	Object seen in region of duodenum—same day	Lost to follow-up	
128906	3	Female	Small screw ½" long	Abdominal pain—not severe	Castor oil, soft food with bread	One day later screw in descending limb of duodenum	Lost to follow-up	
113981	7	Male	Black-headed pin	None	Bulky food	Pin in stomach on same day	Lost to follow-up	
109436	1	Male	Small ring	None	Castor oil, oatmeal gruel and agar-agar	On same day foreign body in antrum of stomach	Lost to follow-up	
108894	2½ yrs.	Male	Pin	None	Castor oil	Pin in region of cæcum (?)	Lost to follow-up	
147786	46	Male	Small plate with 2 teeth	None			Lost to follow-up	
140057	3	Female	Nickle	Slight gagging at time of ingestion. Otherwise negative		In cardia of stomach (½ hour after ingestion)	Lost to follow-up	
148905	3	Female	Whistle	None	None	Whistle in stomach	Lost to follow-up	
151335	3½	Female	Penny	None	None	None	Lost to follow-up	

and stodgy food leaving a residue, such as oatmeal, vegetables, figs, raisins, et cetera, with the addition of a lubricant such as mineral oil, would help to surround the foreign body with a protective coat. When present in the large intestine longer than expected, barium or oil enemata may be used. If symptoms are produced when a foreign body gets to the rectum, it is better to remove it with the finger or proctoscope. Ambulatory treatment is not contra-indicated, although rest is to be desired.

CONCLUSIONS

I. Most foreign bodies of the intestine are ingested accidently and in the majority of cases are evacuated spontaneously regardless of their size, shape, material and number.

II. Trauma from intestinal foreign bodies is guarded against by the protective mechanism of the intestinal wall which produces concavities with muscular boundaries on the mucosal aspect at the points of contact (Exner). This causes an increase in diameter of the intestinal lumen which facilitates propulsion forward of the foreign body by peristalsis and movement of intestinal contents.

III. Foreign bodies, pointed at one end, have a tendency to pass through the intestine with point antiperistaltic and to be evacuated blunt end forward. In this position the point is less likely to impede the progress of the foreign body through the intestinal canal and consequently foreign bodies with blunt end forward will be evacuated more quickly than those with point forward.

IV. A foreign body may travel from the intestine into another organ or into the peritoneal cavity and from there into muscle planes, with little or no symptoms. When late symptoms occur they are referable to the other organ or tissue involved.

V. Conservative treatment of intestinal foreign bodies is indicated in the large majority of cases as shown by statistics and experimental work. Careful observation, rest, and food, or any substance leaving a large intestinal residue may help the successful passage of a foreign body. Cathartics are interdicted.

VI. Obstruction or acute perforation of the intestine or impaction of a foreign body in its wall demands operative therapy.

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THE REPAIR OF INJURIES TO THE POSTERIOR CRUCIAL LIGAMENT OF THE KNEE-JOINT*

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Rupture or stretching of the posterior crucial ligament of the knee-joint is an injury which, until recently, has baffled all efforts to bring about satisfactory repair. In spite of prolonged immobilization of the joint in full extension, or of various operative attempts to suture or reconstruct the ligament, the patient is almost invariably left with a knee in which the femur dislocates violently and painfully forward, whenever weight is borne on the flexed limb. As a rule, such patients are condemned to wearing cumbersome apparatus or to an operation to eliminate motion from the joint.

By far the most important paper on injuries of the crucial ligaments is that of Ernest Hey Groves, of Bristol, published in the *British Journal of Surgery*, 1919–1920. In it he reviews all previous attempts at surgical repair of the ligaments, and describes in detail a new method of operation which he had briefly reported previously (*Lancet*, November 3, 1917), which is brilliant in its conception and in many instances has been highly successful. This operation consists of a free exposure of the interior of the joint by an anterior "U"-shaped incision, the removal of the synovial membrane from the injured ligament and a reconstruction of the ligament by means of a transplant of fascia lata or of the semitendinosus tendon.

A short time after the publication of Hey Grove's preliminary paper, Alwyn Smith (*British Journal of Surgery*, 1918–1919) recommended a modification of the method as applied to the anterior crucial ligament, which substituted a split-patella incision for the extensive reflection of the anterior portion of the capsule of the joint, and added a repair of the internal lateral ligament.

In his second paper Hey Groves (*British Journal of Surgery*, 1919–1920) criticized this modification on the grounds that the incision through the patella did not allow a wide enough view of the interior of the joint and made it impossible to place the new ligament in the exact place of the old one. Without a doubt the approach through the patella does not allow as complete an exposure of all the structures within the joint, but in our experience the exposure is sufficient for the purposes of the operation, and we have found no difficulty in placing the new ligament in the correct anatomical position. We feel, therefore, that any suggestion which will reduce the cutting of ligaments and synovial membrane and lessen the amount of exposure of the interior of the joint to the air should merit favorable consideration, and

* Read before the American Surgical Association, May, 1926.

judging from our own experience and the reports of the results in patients operated on, the method described by Smith is satisfactory.

The operations of Hey Groves and Alwyn Smith were nearly all performed for injury of the anterior crucial ligament which is much more frequently injured than the posterior. In these operations a new ligament, fashioned from a transplant of fascia lata or ileotibial band, was passed through drill holes in the tibia and the femur, which were so placed that the new ligament traversed the cavity of the joint somewhat in the position of the injured ligament. When healing occurred between the transplant and the tunnel through the bone the new ligament assumed more or less perfectly the function of the injured crucial, and so prevented the tendency to subluxation. In the case of injuries of the posterior crucial, however, Hey Groves neglected to bury the posterior end of the new ligament in the tibia, and in failing to do so would appear to invite failure of the operation. In the method which he describes the tendons of the semitendinosus and the gracilis are cut free from the muscle in the middle of the thigh, and the cut ends are drawn into the interior of the knee-joint by a pair of clamps, which are thrust from before backward through the posterior ligament. These tendons are then passed through a hole drilled through the internal condyle at the point of the anterior attachment of the posterior crucial and drawn taut. Unfortunately the new ligament has no secure anchorage to the tibia, except in so far as the tendons heal to the hole in the posterior ligament of the knee-joint, and consequently can have very little strength to resist the tendency of the femur to dislocate forward on the tibia. This appeals to us as a defect in the method which might readily be remedied.

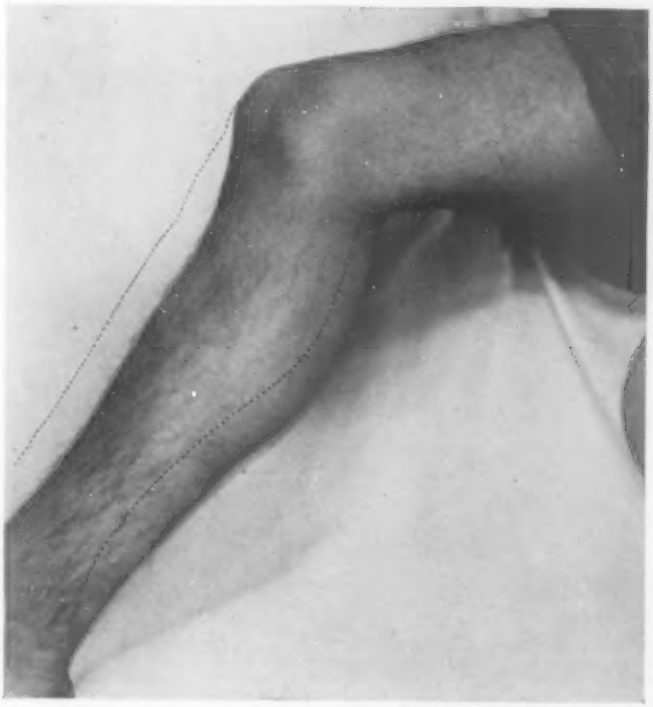

FIG. 1.—Photograph of the knee of the patient described in text in which the posterior crucial ligament had been ruptured. It shows the subluxation of the femur forward on the tibia. The dotted lines show approximately the normal relation of the leg to the thigh.

While recognizing, therefore, that to Hey Groves all honor is due for the

conception of the idea that the crucial ligaments might be replaced by transplants of the fibrous tissues, and for showing that the idea was feasible by practical demonstration on patients, yet we wish to point out certain features of his operation which appeal to us as amenable to improvement: (1) The approach to the joint by way of an incision which passes transversely through the tubercle of the tibia and across the whole of the anterior portion of the capsular ligament appears to be a rough way of dealing with such an important joint and one which may lead to laxity of the ligaments when healing is complete. (2) The damage to the synovial membrane caused by the primary incision and by the dissection necessary to expose the crucial ligaments is likely to result in synovitis and subsequent chronic arthritis. (3) The prolonged exposure of the interior of the joint adds to the risk of infection. (4) The placing of the transplant in the interior of the joint in such a way that its surfaces are uncovered by synovial membrane, and are bathed in synovial fluid instead of lymph, introduces a modification of the accepted method of transplanting fibrous tissues which, while it may be perfectly satisfactory, may result in pathologic changes which will lessen the efficiency of the operation. (5) In the case of the operation for rupture of the posterior crucial ligament the posterior attachment of the new ligament is not sufficiently secure.

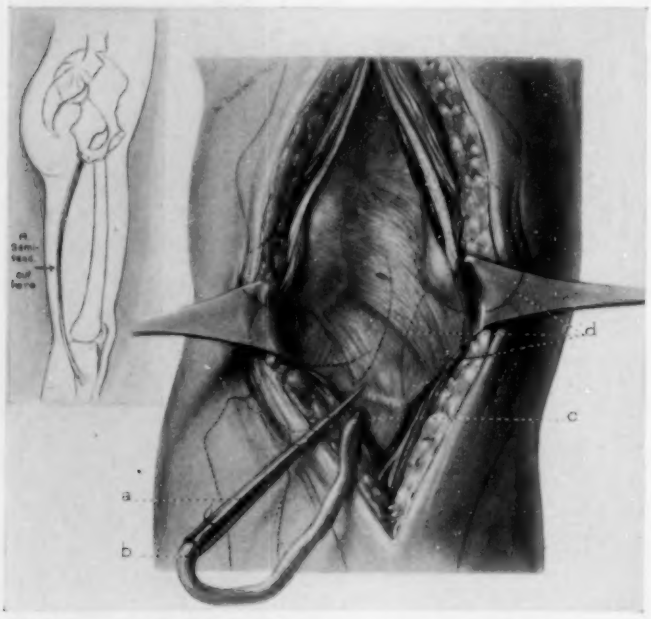

FIG. 2.—Drawing designed to illustrate the steps of the operation through the posterior incision. The tendon of the semitendinosis has been detached from the muscle high in the thigh and freed down to its insertion into the internal surface of the head of the tibia. It has then been passed from before backward through a drill-hole through the head of the tibia and is ready for introduction into the joint. (*a*) Sharp-pointed bodkin, with the assistance of which the tendon has been passed through the head of the tibia, and which is now in position to be thrust through the posterior capsule and along the remains of the posterior crucial ligament without injuring the membrane; (*b*) tendon of semitendinosis; (*c*) drill-hole through head of tibia; (*d*) dotted lines showing the outlines of the bones and the posterior crucial ligament in the interior of the joint.

The suggestions which we have to offer were tried out first in the anatomical department and later on a patient who had sustained a rupture of the posterior crucial ligament. They substitute for the extensive transverse incision the vertical split-patella incision or a vertical incision just internal to the patella. The use of such an incision removes the danger of increasing

the laxity of the capsule which attends the transverse incision and does infinitely less damage to the synovial membrane. Further, they reduce the time that the interior of the joint must be exposed, both by lessening the size of the incision and by eliminating the removal of the synovial membrane from the ligaments. Although the whole operation must be considered a prolonged one, the actual time during which the joint was open in the case we shall describe was not more than ten minutes. Again, the new ligament is introduced in such a way that it is entirely subsynovial. By this arrangement the transplant is placed in the exact anatomical position of the injured ligament and is left clothed by the synovial membrane which normally covers it. In this way the new ligament is buried throughout in vascular tissue, with no portion of its surface exposed in the joint or to the synovial fluid. The absence of practically all interference with the synovial membrane reduces to a minimum the post-operative inflammatory reaction and the possibility of adhesions or scars within the joint. And finally, a solid anchorage is provided for the new ligament, not only to the femur in front but also to the tibia behind, so that the reconstruction of the ligament is, as far as we can see anatomically perfect.

The patient was a young man, aged twenty-seven years, who six months previously had been struck down by a falling tree which fell across the outer side of his flexed knee in such a way as to rupture or stretch the internal lateral ligament and force the head of the tibia backward. The limb was immobilized in full extension for two months, until the hæmarthrosis and subsequent synovitis had disappeared, and he then began to walk. He was unable to return to work, however, owing to the circumstance that whenever weight was borne on the knee in any position except full extension the femur dislocated violently and painfully forward on the tibia for a full inch and the limb as a support became useless (Fig. 1). In addition, there was considerable lateral mobility of the joint even when in full extension, owing to faulty repair of the internal lateral ligament.

The operation performed consisted of several distinct steps (Figs. 2 and 3):

1. Through a long medial incision, which extended from the middle of the back of the thigh to the upper portion of the calf, the tendon of the semitendinosus was exposed and isolated. It was then detached from the muscle as high as possible in the thigh and stripped downward toward its insertion.

2. By way of the superficial portion of the split-patella incision the insertion of the semitendinosus was exposed on the inner surface of the upper end of the tibia and the whole tendon drawn through to the front of the leg from beneath the sartorius.

3. The lower portion of the posterior medial incision was then deepened and the space between the gastrocnemii muscles located and widened until the posterior ligament of the knee-joint and the upper portion of the posterior

surface of the head of the tibia came into view. The vessels and nerves were retracted to the outer side. A ¼-inch drill was applied to the posterior surface of the head of the tibia at or slighty external to the midline and close up to the attachment of the posterior ligament to the bone. The drill was so inclined that it emerged on the internal surface of the tibia close to the insertion of the semitendinosus. The cut end of this tendon was then fastened into the butt end of a sharp-pointed bodkin, and by using the bodkin as a needle the tendon was passed through the head of the tibia until it appeared in the posterior incision.

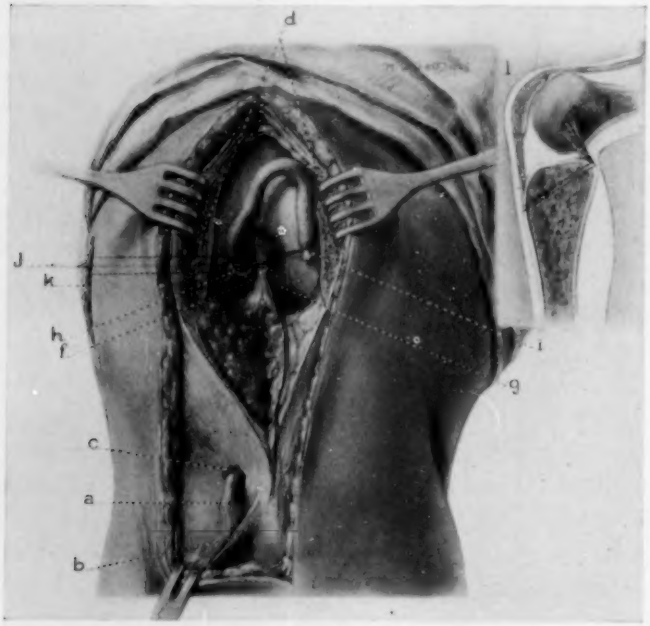

FIG. 3.—Drawing designed to illustrate the steps of the operation through the anterior incision. In the lower portion of the incision the semitendinosis tendon can be seen at its insertion subjacent to the sartorius, which has been retracted. It has been passed from before backward through the hole in the head of the tibia, and, with the knee flexed and the patella split and retracted, the bodkin shown in Fig. 2 has been pushed forward through the posterior capsule and along the posterior crucial ligament until the point punctured the synovial membrane at the most anterior point of attachment of the posterior crucial ligament to the femur. The tendon was then drawn into the joint, the bodkin detached and the end of the tendon fastened to a flexible wire-threader which has been passed through a hole drilled through the internal condyle of the femur in such a direction as to emerge in the joint just subjacent to the punctured hole in the synovial membrane covering the attachment of the posterior crucial ligament to the femur. The tendon will now be drawn through the internal condyle and will disappear beneath the synovial membrane. (*a*) Insertion of semitendinosis tendon; (*b*) insertion of sartorius; (*c*) drill-hole through which the semitendinosis has been passed through the head of the tibia to appear at the back of the joint; (*d*) lateral halves of the patella retracted; (*f*) posterior crucial ligament; (*g*) anterior crucial ligament; (*h*) puncture hole in synovial membrane covering attachment of posterior crucial ligament to the femur; (*i*) tendon of semitendinosis from which the bodkin has been detached and to which a silk ligature has been fastened preparatory to drawing it through the hole in the internal condyle of the femur; (*j*) wire-threader passed through hole in internal condyle which is indicated by dotted lines; (*k*) small incision over most prominent point of internal condyle of femur; (*l*) drawing of lateral surface of the medial half of the knee-joint which has been sectioned in the sagittal plane. It shows the tunnel through the head of the tibia and the line of the new ligament in the correct anatomical position.

4. The patella and patellar tendon were split longitudinally and the interior of the joint exposed. With the patient lying on his back and the knee flexed over the end of the table, the operator placed himself so as to look into the joint. The sharp-pointed bodkin was then pushed from behind forward through the posterior ligament of the knee at a point just above the hole in the head of the tibia and in the line of the posterior crucial ligament. As soon as the point of the bodkin had passed through the posterior ligament the operator could detect its presence within the synovial sheath of the posterior crucial ligament by wiggling the point about. The bodkin was then pushed forward until its point punctured the synovial membrane at the most anterior point of the attachment

of the posterior crucial ligament to the internal condyle of the femur. The bodkin and tendon were finally drawn out through the split-patella incision.

5. A small incision was now made over the subcutaneous portion of the internal condyle of the femur and the drill passed through this bone so as to enter the joint at the exact spot at which the bodkin had punctured the synovial membrane over the insertion of the crucial ligament. The bodkin was next detached from the tendon and the tendon fastened to the eye of a flexible wire-threader which had been passed into the joint through the hole in the internal condyle. The end of the tendon was then drawn through the internal condyle, and when it was drawn taut the new ligament was seen to disappear through the small puncture wound in the synovial membrane and become entirely subsynovial.

FIG. 4.—Photograph of patient described in text, six months after the operation. In this position the knee invariably dislocated previous to operation. Compare with Fig. 1.

6. The knee-joint was now placed in full extension, the tendon drawn taut and its terminal end sewn down to the internal lateral ligament. The wounds were closed with catgut and horsehair and a plaster bandage applied from the toes to the top of the thigh.

Two months later the plaster was removed and the patient allowed to exercise the knee gently. At first there was not more than 20 degrees of motion, but within a week this had increased to 100 degrees. A slight synovial effusion appeared within twenty-four hours of the removal of the plaster, but this had disappeared in a week. The patient then began to walk.

Nearly three years have now elapsed since the operation. The patient

has been examined from time to time and has made steady improvement. He has worked as a chauffeur, as a waiter and as an ordinary laborer. He walks with a normal gait and has a complete range of motion in the joint. The previous tendency to subluxation has been cured and the new crucial ligament performs perfectly the functions of the old. His only complaint is that the joint is a little weak and the physical examination shows that this complaint is justified as he has more than the normal degree of lateral, rotary and antero-posterior mobility, sufficient to cause the knee to tire after prolonged heavy labor. The result as shown in (Fig. 4), however, is a great improvement on the original condition.

One feature of the operation for the repair of the posterior crucial ligament, which we think is applicable to the anterior crucial ligament as well, is the introduction of the new ligament in such a way that it is entirely subsynovial. On the only occasion, however, on which we have done a reconstruction of the anterior crucial ligament, the ligament and the synovial membrane covering it had been completely torn across and there was not sufficient synovial membrane left to cover the transplant. Judging from the description of the interior of the joints in cases that have been reported, it is unusual to find any evidence of injury to the synovial membrane, so that we intend when the occasion arises to make the new ligament subsynovial here also. We have done it on the cadaver and it can be accomplished with only two small slits in the synovial membrane, as it covers the old ligament at its points of attachment to the femur and tibia. When the transplant is drawn into position these slits close spontaneously and the new ligament disappears from view.

The question of the permanence of ligaments made from tendons or fascia is one that can only be solved by time. There is no doubt that if such tissues are transplanted from one place to another in the same individual, and if they are placed in contact with a good supply of lymph, they will continue to live unchanged. We have demonstrated, however, that in order that the whole thickness of a transplanted tendon may live, it is necessary that it be split open so that lymph may reach its interior. Ten years ago we described a method by which the tendons of paralyzed muscles could be converted into ligaments, and so prevent deformities of the feet, by burying them in troughs in the bone (ANNALS OF SURGERY, October, 1915). These new ligaments are undoubtedly permanent. In order that the adhesion of the transplanted tendon to the bone may be sufficiently strong to overcome the tendency to deformity, however, it is necessary to bury it in a tunnel or trough, and it is essential to remove all areolar tissue and to scarify its surfaces thoroughly. If these precautions are taken the fixation of the transplant to the bone will be strong and permanent. In the case of the crucial ligaments, therefore, if a stout transplant of fascia lata or tendon be used, such as will fit snugly into a ¼-inch drill hole, and if the precautions necessary to successful tendon fixations are observed, we believe that the immediate successful results will stand the test of time.

TRANSACTIONS

OF THE

NEW YORK SURGICAL SOCIETY

Stated Meeting Held December 8, 1926

The Vice President, Dr. Frank S. Mathews, in the Chair

I. ADENOMA OF THYROID—MALIGNANCY—PERFORATION OF PLEURA

Dr. Charles Gordon Heyd presented a woman, age fifty-three years, who entered the New York Post-Graduate Medical School and Hospital, October 26, 1926, complaining of swelling of the neck, duration forty years. When the patient was thirteen years of age she noticed a swelling on the right side of the neck, which at that time was diagnosed as "goitre." This tumor remained quiescent for thirty-five years when it gradually but continuously increased in size. About four months ago the patient noticed a small "lump" on the left side of neck which has grown very rapidly. Coincident with the increase in the size of the left-sided tumor the patient has had pain in the throat, with choking sensations, difficulty in swallowing, dry cough and increasing dyspnœa. In addition, during the last four months the patient has lost twenty pounds in weight but does not believe this is due to the difficulty in swallowing.

On physical examination the patient is obese, with evident dyspnœa and difficulty in swallowing, together with a hoarseness of the voice. Examination of the neck showed an adenoma of the right lobe of the thyroid approximately 12 cm. in length. On the left side there was another tumor approximately 10 cm. in length, extending out laterally and passing down beneath the left clavicle. There were no signs of hyperthyroidism; pulse rate 70; no tremor; no eye signs. Röntgenological examination demonstrated a retrosternal goitre and examination of the larynx revealed limited motion of the left cord, with the cord in abduction simulating a cadaveric vocal cord.

Operation was performed, October 27, 1926, under rectal-ether anæsthesia. As a preliminary safety measure an intertracheal breathing tube was inserted into the trachea. The operative findings revealed a large retrosternal mass evidently originating on the left side. It was hard and fixed to the surrounding muscular tissue and passed outward and downward beneath the carotid artery and jugular vein. Grossly the tumor strongly suggested an infiltrating carcinoma. On the right side was a somewhat smaller mass, much softer in consistency, passing downward beneath the clavicle but not encroaching on the midline and lying anterior to the carotid artery. There was no gross external evidence of malignancy in this mass. It suggested only a simple encapsulated adenoma. The operation consisted of a typical exposure for a thyroidectomy. Thyroid resection was begun on the right side with division of the superior pole and the anterior four-fifths of the right lobe were resected from above downward and the mass delivered easily without hemorrhage from below the clavicle. On the left side by reason of the perforation of the capsule of the thyroid there was deep penetration into the surrounding muscles. The mass was fixed posteriorly and was beneath the carotid artery, jugular vein and vagus nerve. It was densely adherent anteriorly to the carotid sheath. Resection was begun at the superior pole and the carotid

artery and jugular vein were separated from the mass by scalpel dissection. The lower pole of this mass extended well beneath the sternum and the clavicle on the left side and was apparently attached to the dome of the pleura in the left supraclavicular space. Upon turning this mass downward and forward on the chest it was rotated from the retrosternal space and delivered. Following this there was an escape of air. Examination of the trachea revealed no defect, and although the laryngeal nerve was exposed in the dissection, it was left untouched. Deep in the supraclavicular space air would bubble up upon the removal of gauze pressure. This cavity was packed lightly with iodoform gauze and wound closed in anatomical fashion. About one-fifth of the thyroid was left on the right side while a complete thyroidectomy was performed on the left side.

Pathological examination showed that we had two distinct tumors from this patient, the one on the right being soft and the one on the left dense and hard. In brief there were two large nodular ovoid masses, one of which was soft and the other firm. The soft mass measured 11 x 5.5 x 5 cm. The firmer of the two masses measured 8.5 x 6 x 6 cm. It was imperfectly encapsulated and on section the cut surface was moist, pale gray mottled and streaked with bright red and various shades of yellow. Microscopically the soft tumor consisted of large irregular alveoli of eosinophilic epithelial cells provided with rounded nuclei and occasionally showing mitotic division. The cells were often vacuolated and there was a tendency for the formation of spaces between adjoining cells, these spaces being filled with colloid. There was very little stroma and the tumor alveoli were intimately attached to wide thin-walled blood-vessels. In striking contrast with this picture the hard tumor mass showed occasional thin-walled thyroid alveoli in a densely fibrous matrix which contained spindle-shaped, irregularly stellate cells of very bizarre appearance. These cells were basic staining with reticulated cytoplasm and large irregular nuclei. Their number as well as their size varied greatly in different places in this scirrhous growth. Mitotic figures were fairly numerous and multiple metastases were observed. Areas of necrosis occurred in this part of the growth. Pathological diagnosis, adenocarcinoma of thyroid, right; scirrhous carcinoma of thyroid, left.

On the evening of the day of operation patient had a well-defined emphysema over the anterior chest and neck. This gradually subsided and at the end of thirty-six hours had completely disappeared.

The gauze packing in the supra-clavicular fossa was gradually removed and was entirely out of the wound on the fifth day. Patient made an uneventful recovery and was discharged, November 13, 1926, eighteen days after operation.

It is interesting to note that the left-sided mass apparently had its origin from the right, yet on the right side there was a typical adenocarcinoma and on the left side a scirrhous carcinoma. From an anatomical point of view it is interesting also to note that the left-sided mass passed outward and downward beneath the carotid artery and jugular vein, a very unusual anatomical finding.

II. ADENOMA OF THYROID—STRUMA NODOSUM—MALIGNANT

Doctor Heyd also presented a woman, age thirty-five years, single, stenographer, who entered the New York Post-Graduate Medical School and Hospital, October 29, 1926, complaining of goitre of five years' duration. The patient states that about five years ago she began to notice an enlargement in the right side of neck in the region of the thyroid gland. This has very slowly increased in size and is entirely without symptoms. On physical examination there is a glandular mass at the site of the right thyroid gland,

freely movable, about five cm. in diameter. Operation was performed October 29, 1926, under ethylene gas anæsthesia and a typical resection of the right lobe of thyroid and portion of isthmus carried out. On gross examination there are two pieces of thyroid tissue, one flattened and roughly elliptical in form measures 5 x 3.2 x .15 cm. The other is a lobulated mass one end formed by a soft ovoid nodule measuring 5.2 x 3.2 x 3 cm. To the centre of this is attached a small flattened mass of tissue 3.5 x 3 x 1 cm. The combined weight is 47 grams. The smaller piece of tissue on section shows reddish fleshy thyroid tissue in which are embedded projecting yellowish-gray, semi-translucent nodules 1 cm. in diameter. The centre of one of these nodules has been reduced to an irregular shaggy cyst. Section of the larger mass shows the ovoid lobe to be composed of two definitely encapsulated tumors. One measuring 3.2 cm. in diameter, bulges above the cut surface, is soft grayish-yellow and mottled with red. Above this is a somewhat smaller tumor with central cystic softening. In the adjoining mass of thyroid tissue is a less definitely demarcated lobular tumor nodule 2 cm. in diameter.

Microscopically sections from various portions of the tissue show a moderately variable structure. Everywhere there are colloid-containing alveoli in the thyroid lobules, but these alveoli are quite variable in size. Many of them are very small. Their lining epithelium consists of rather tall cells and in the lining epithelium there are occasional distinct mitotic division figures. Many of the alveoli are solid without colloid. The colloid itself is vacuolated and stains rather poorly. At the capsule one sees solid groups of epithelial cells extending into the fibrous tissue. These groups appear compressed, but nevertheless the cells composing them are relatively large and their cytoplasm takes faint tinge of hematoxylin. There are numerous lymphocytes in the connective tissue not only of the interlobular septa, but also abundant within the lobules themselves. In one portion the stroma is very œdematous with well-defined thyroid alveoli scattered in it. The rapid growth is indicated by the presence of mitotic division figures, the abnormal staining of the epithelial cells and the poorly formed colloid as well as the presence of numerous collections of lymphocytes indicate a very profound disturbance of structure in which growth activity of the epithelial cells appears to be the essential feature. The transformation appears not to warrant an unqualified diagnosis of carcinoma, but at the same time the growth activity indicates a definite malignant tendency. Pathological diagnosis, multiple malignant adenomata.

The post-operative convalescence was uninterrupted and patient was discharged from the hospital, December 4, 1926, six days after operation.

Comment.—This patient had no signs of hyperthyroidism and had simply a freely movable adenoma, yet the histological examination revealed multiple malignant adenomata, the diagnosis being made upon the marked cellular metaplasia with the high degree of mitoses of the specimen.

III. ADENOMA OF THYROID—STRUMA NODOSUM—BENIGN

Doctor Heyd also presented a woman, age forty-three years, married, who entered the Post-Graduate Medical School and Hospital, November 9, 1926, complaining of swelling of the neck, tachycardia, nervousness and weakness. About eighteen years ago she noticed a swelling in the neck which has gradually increased in size up to date. About two years ago noticed an increasing frequency of pulse rate and a general weakness, but without any loss of weight. She has also complained of increasing nervousness without any apparent cause. The patient has had three basal metabolism

tests, all of them plus, with an average of plus 24. On physical examination the patient is obese, with an irregular enlargement and deformity of the thyroid gland. The pre-operative diagnosis was adenomata of thyroid, with moderate hyperthyroidism. There was no history of iodine administration. The patient was operated upon on November 10, 1926, under ethylene gas anæsthesia. Operation findings were: The thyroid gland is entirely replaced by multiple adenomata involving the both lobes and isthmus. Many of the adenomata are degenerated into cystic hemorrhagic masses. The right lobe is approximately 10 x 7 x 10 cm. The left is 15 cm. in length, 10 in depth and about 7 in transverse diameter. The left side is completely substernal with pressure upon the trachea and adjacent soft parts. Operation consisted of typical collar exposure with resection of the right lobe from above downward, leaving about one-fifth of the degenerative tissue at the superior pole together with a small remnant of thyroid tissue on the posterior capsule. A bridge of fairly normal thyroid tissue was left across the trachea. On the left side the tumor mass was resected from above downward and about seven-eighths of entire lobe and adenomatous tissue removed. Hæmostasis was assured and the wound closed in anatomical fashion without drainage. Microscopically the sections from various parts of the tissue showed considerable variation in structure. In some places there were thyroid lobules made up of colloid-containing alveoli fairly regular in size, but in these portions there were collections of lymphocytes in the fibrous stroma and there was an evident increase of connective tissue, especially around the lobules. All but the smallest alveoli contain colloid in these portions. The lining epithelium was low columnar or cuboidal in type. In some places the fibrous septa were one-half to one mm. in thickness. Sections of one piece showed a much more marked alteration of structure. Here the epithelial cells formed very small alveoli without colloid or occurred as small clumps of cells in an œdematous stroma. Well-formed thyroid alveoli were not found in this portion at all. It presented a thin fibrous capsule at the periphery which appeared to be respected everywhere by the epithelium. A brief search for mitotic division figures was negative. Pathological diagnosis, multiple adenomata of thyroid gland with one quite atypical adenoma, the structure of which suggests pre-malignant tendency.

Post-operative convalescence was normal. Patient was discharged from the hospital, November 21, 1926, twelve days after operation.

Comment.—The histological examination here showed a condition of multiple adenomata of the thyroid with cellular metaplasia but an absence of mitoses. It is interesting to speculate whether the type of thyroid histology had been influenced by the co-existing hyperthyroidism.

IV. ADENOMA OF THYROID—PERFORATION OF ŒSOPHAGUS

Doctor Heyd also presented a woman, age sixty years, who entered the New York Post-Graduate Medical School and Hospital, April 13, 1921, complaining of tumor of the neck. For the previous ten years the patient had noticed a gradual increase in the size of her neck. This was unaccompanied by symptoms up to three months before she entered the hospital, when she began to have some difficulty in breathing and swallowing and a sensation of constriction in the neck. On physical examination the patient showed a cystic type of goitre, more pronounced on the left side, extending beneath the clavicle. She was operated upon April 14, 1921, under ether anæsthesia and a typical bilateral resection performed. On the left side the tumor was firmly adherent to the trachea and to the muscular tissues behind the thyroid cartilage. During the operative procedures it was not apparent

that there was any perforation in the œsophagus. Thirty-six hours after operation patient had a temperature of 104.8° and pulse of 150. Under the impression that we had a condition of post-operative hyperthyroidism, the dressing was taken down and the wound partially opened with the escape of about thirty cubic cm. of thin, yellowish fluid. A rubber tube drain was then inserted and at the end of forty-eight hours the character of the discharge assumed a more purulent appearance. On the fourth day water taken by mouth passed out the goitre wound. A duodenal tube was inserted and everything by mouth interdicted. The patient was fed entirely through the duodenal tube for eight days, or from the fifth day to the thirteenth day after her operation. During this period the cervical discharge was diminished to a small amount of mucoid material. The temperature became normal on the ninth day after operation and the patient was allowed up in a chair on the tenth day. During the eight days that the duodenal tube was in place the patient had no disturbance except a slight complaint of "phlegm" in the throat. At the end of eight days the duodenal tube was withdrawn, but it was found that the patient still had an œsophageal fistula because on drinking water a small amount came through the wound. The duodenal tube was reinserted and left in place until the twenty-third day after operation or an additional eight days when the tube was withdrawn and the patient was fed by mouth. The œsophageal fistula remained permanently closed. The patient was discharged from the hospital twenty-seven days after operation and except for a slightly increased scar at the original point of thyroid drainage revealed nothing noteworthy as the result of the œsophageal leakage. From the fourth day after operation until the twenty-third day after operation the patient had nothing by mouth and in all the duodenal tube in place sixteen days with an interruption of one day but without oral feeding. The disturbance from the duodenal tube was slight and a rather marked œsophageal fistula closed spontaneously in sixteen days. The patient's nutrition suffered some depreciation during the régime of duodenal feeding as the amount of nutrition and the variety of dietary articles that could be introduced through the duodenal tube are distinctly limited.

Comment.—This patient has remained perfectly well to date.

V. BONE METASTASES FROM ADENOCARCINOMA OF THE THYROID, SIX YEARS AFTER THYROIDECTOMY

Doctor Heyd also reported a case of adenocarcinoma of the thyroid operated upon June 2, 1919, at the New York Post-Graduate Medical School and Hospital. The patient was a man, at that time fifty-five years of age, who was seen in consultation for marked dyspnœa. The patient had been complaining of enlargement of the thyroid for the previous five years, the last three of which had been associated with a progressive and continuous difficulty in breathing. For the last year had noticed a change in his voice and at the time of examination could speak only in a whisper. In addition, the patient had a marked hacking cough throughout most of the day and night. On examination the patient showed a bilateral enlargement of the thyroid with mass extending down beneath the sternum. X-ray examination revealed a large retrosternal goitre. Under ether anæsthesia on June 2, 1919, a complete thyroidectomy was performed. Very little technical difficulty was encountered in removing the substernal mass, 10 x 5 x 6 cm., and weighing 218 grams. The patient made an uneventful recovery and was discharged on the eleventh day after operation. Microscopically the tumor consisted of closely packed columns of epithelial cells without colloid and with only very slender threads of connective tissue between them. Such closely packed

columns made up large areas 2 to 10 mm. in diameter of practically solid epithelial tissue. Mitotic figures were present in the epithelial cells, and, in places, necrosis and hemorrhage. Pathological diagnosis, thyroid adenocarcinoma.

After leaving the hospital the patient, during the next year, submitted to three complete series of X-ray treatments and regained his weight, and was able to return to his duties as Inspector of Police, and aside from a permanent hoarseness was in every way clinically cured. The patient enjoyed uninterrupted good health until September, 1926, when he returned complaining of pain in the lower extremities, œdema of both legs, loss of weight and a tumor in the right thigh, upper third, inner side. His history at this time was that two years ago he noticed a small lump on the inner side of the right thigh and began to complain of weakness of the legs, with pain, particularly on the right side. At this time sugar was found in his urine and he was treated intermittently for glycosuria. On examination the patient was cachectic and appeared an extremely ill man. To the inner side of the right femur was a mass the size of a fœtal head at term, somewhat movable and did not appear attached to the bone. On rectal examination a mass was felt to the right of the rectum occupying the true pelvis. The diagnosis at this time was vertebral and spinal metastases with pelvic tumor, secondary to adenocarcinoma of the thyroid. X-ray examination by Doctor Meyer at the Post-Graduate Hospital on October 1, 1926, showed areas of destruction involving the inner margin of the brim of the true pelvis on the right side with further metastatic like areas in the right pubic bone and ramus extending toward the ischium, with a further small focus of rarefaction in the left pubic bone. Further evidence of metastasis also appeared to exist in the left femur below the trochanters. Röntgenological diagnosis, metastatic malignancy.

Comment.—At the operation there was nothing about this tumor to suggest malignancy unless it were the unusual hardness and density of the mass. Following the operation the patient was submitted to rather intensive and extensive X-ray therapy. He enjoyed almost uninterrupted good health for seven years, the last two years of which, however, were undoubtedly characterized by the development of his vertebral and pelvic metastases. At the present time he is being treated palliatively with X-ray for his pain.

Dr. Howard Lilienthal referred to Doctor Heyd's second patient as to whether or not the patient should be told that the growth he had removed from her neck was malignant. He thought that it was not necessary to give this information to the patient herself but that her family should have it. He believed that many of these malignant tumors of the thyroid are slow to recur and that this one might not recur for many years. In one of his first cases of cancer of the thyroid he removed what was apparently an adenoma; it was later reported from the laboratory to be an adenocarcinoma and the patient was sent home to die. Three years afterward he was perfectly well. Another case, also a malignant adenoma of the thyroid with extirpation and subsequent myxœdema apparently made a perfect recovery; he had followed this case for at least ten years. When the patient stopped taking thyroid during an acute illness, there was an acute return of the symptoms of myxœdema, but they disappeared on the resumption of the thyroid therapy. Doctor Lilienthal said he did not think cancer in any other part of the body would give so much hope that the patient might go on for years without recur-

rence, as that of the thyroid. In another case of malignant adenoma of the thyroid, on which he had operated, he removed only the diseased part. The patient apparently recovered but later had recurrence below the jaw. The case went on with X-ray treatments for six years and then the patient died. Many of these cases are of extremely slow malignancy, and Doctor Lilienthal expressed the hope that this young lady would never have any further trouble with her thyroid.

Dr. John Rogers said that he had operated on many of these malignant tumors of the thyroid and he believed it difficult to make a diagnosis beforehand. When a tumor of the thyroid has the appearance of an adenoma, but can be enucleated without rupturing the capsule, the prognosis is extremely good. He believed the prognosis in the case of this young woman presented by Doctor Heyd was good.

Dr. Walter A. Sherwood said he had observed a case on which he had operated on the assumption that it was simple adenoma. A very incomplete operation was done, merely a resection of one lobe of the gland, but the laboratory reported malignancy. This was two years ago. The man's toxic symptoms disappeared after the operation and the remainder of the thyroid has decreased in size and there is no sign of recurrence. In view of this Doctor Sherwood felt he could substantiate the opinions of Doctor Lilienthal and Doctor Rogers.

In closing, Doctor Heyd said he was moved to present these cases of adenoma to emphasize the inherent danger of leaving adenomata of the thyroid unremoved. There is a well-evidenced tendency for some of them to show changes that lead to grave hyperthyroidism. Again, the continued presence of an adenoma with increasing growth induced sooner or later some degree of pressure symptoms and, finally, all adenoma were potentially malignant. The fact that the first case had carried a tumor on the right side of her neck for forty years and then had a malignancy develop was proof that an adenoma, left sufficiently long, had in it the possibilities of cellular metaplasia leading to malignancy. In the second case—struma nodosa malignum—Doctor Heyd was not prepared to inform the patient that she had a malignant goitre, nor would he submit the patient to X-ray or radium therapy because the adenoma being well encapsulated offered a reasonably good prognosis. However, he agreed with Doctor Lilienthal that some member of the family should be apprised of the possibilities of later manifestation of malignancy.

ACUTE POST-OPERATIVE HYPERTHYROIDISM

Dr. Harold E. Santee presented a young woman, age nineteen years, who was referred to Bellevue, Second Surgical Division, by Dr. John Rogers, October 5, 1926. She gave a history of increasing nervousness, insomnia, vasomotor instability as indicated by flushing and sweating, from July, 1925, to January, 1926. At this time, enlargement of the thyroid was apparent. Severe headaches, tremor, tachycardia, progressive weakness and loss of weight were apparent in spite of increased appetite and food consumption, and from January to June, 1926, most of her time was spent in rest at home and in bed in an effort to overcome her trouble. Palpitation, tremor, extreme

nervousness, precordial pounding of the heart, diarrhœa, weakness and a loss of weight of twenty-five pounds in twelve months briefly describe her condition during the summer. Exophthalmos was moderate and the thyroid was diffusely and greatly enlarged with all the local characteristics of a true hyperplastic gland. Under a very careful régime during the month prior to admission to Bellevue, some weight was regained but the exact amount was uncertain.

The excitement of hospitalization increased her pulse rate to 140 and on October 7, 1926, her basal metabolism was plus 62 per cent. Lugol's solution, 10 mms., t.i.d., was given with obvious benefit subjectively and with considerable benefit objectively for the following nine days. Her metabolism on October 14, 1926, was plus 48 per cent. Although clinical improvement was fairly marked, this particular case as an operative risk was viewed with apprehension by the reporter; not so much because of any laboratory test as by reason of that intangible feeling or "hunch" that is apt to result from a close observation of an individual case of Graves' disease. Perhaps the main factor in this was a close observation of the soft fulness of the pulse and its irritability in response to mild mental and physical stimuli.

For this reason, a ligation of both inferior thyroid arteries under local anæsthesia was done on October 16, 1926, not for any therapeutic effect, but as an aid to later resection. No undue reaction resulted and Lugol's was resumed after three days. On November 6, under gas, oxygen anæsthesia, a resection of both lobes was done, excising about three-fourths of each lobe. The gross findings and the microscopic examination were characteristic of Graves' disease. The procedure, however, was too long for this individual and the operator is frank to say that he overstepped the threshold of safety in this case. This was realized, however, and in addition to the usual post-operative medication of these cases, three doses of an aqueous thyroid extract were given intramuscularly, mms. 20, q.2.h. Bromides in rectal tap and morphine were also used. Approximately twelve hours after operation, the temperature began to rise and she grew gradually more and more restless. Sixteen hours after operation, the temperature had risen to 107 degrees. Quoting from the report of the House Surgeon (Doctor Heim), who stood by the patient through the night hours: "Patient was in a wild, delirious, uncontrollable state. Pulse was so rapid it could not be counted. Every measure possible was administered to bring down temperature; sponge baths, ice packs, exposure to draughts. A total of a grain of morphine had been given in twelve hours which seemed to have no effect. The most dramatic effect was then observed after the administration of 30 minims of aqueous thyroid extract intravenously. Five minutes after this administration, patient had changed from a raving maniac to complete quietness. Pulse had dropped to within countable limits and was around 120. Quality had completely changed. Whereas before it was extremely rapid, thready, and weak, now it was full, bounding and strong." Three such intravenous doses were given at two-hour intervals. Temperature dropped to around 102°. A second exacerbation of toxicity occurred twelve hours later which was also alleviated by similar dosage, following which the patient was continued on intramuscular dosage of fifteen minims twice a day for three days. The neck wound was infected, probably due to the extreme muscular activity and sweating combined with the various therapeutic measures used at the height of her acute toxæmia. This, however, subsided gradually and following the first three days post-operative, the patient progressed well until her discharge from the hospital, November 21.

This case was shown as illustrating the use of what seems to be an

effective therapeutic agent in that occasional tragic sequence to operation in this disease. Two months ago, Dr. John Rogers reported in *Surgery, Gynæcology and Obstetrics,* similar striking effects in three cases. Personally, the reporter had seen three cases in which he feels that life has been saved by the use of this aqueous thyroid extract. The agent itself is an aqueous extract of fresh pig thyroid from which all coagulable material has been removed and the resulting extract concentrated down to contain a definite amount of iodine to the cubic centimetre. Its employment rationally seems to be explained on the grounds that it supplies a farily normal utilizable thyroid extract to a patient suffering from either a complete suppression of the normal secretion or a highly toxic secretion, perhaps both. In the cases in which he had used it, no toxic effects have ever been noted. However, it does not work in every case but is to be considered a possible measure of relief in those tragic cases like the above.

Dr. John Rogers said this case was a very interesting illustration of the probable pathologic physiology which underlies these disturbances. One cannot think of them as the result purely of an excess of toxic secretion in the thyroid. At autopsy these thyroid glands are found to be almost solid with practically no colloid in the alveoli. Instead the alveoli are represented by disintegrating masses of cell tissue. It is hard to believe the gland has secreted anything like normally. These cases of toxæmias occur quite rapidly. The speaker had been successful in giving the "thyroid residue" in many of these post-operative toxæmia cases and he found it a valuable thing to have at hand. Many of these cases become suddenly alarmingly ill during the operation, and an injection of the thyroid residue (20 minims) will slow and strengthen the heart action.

Going back to the pathologic physiology. It is well known that the feeding of any thyroid product increases the blood sugar. The acute toxæmia and chronic toxæmia can both be relieved by glucose in a solution of 50 per cent. It has to be chemically pure and can be given in post-operative toxæmias once in twenty-four hours. This glucose seems to act beneficially in all the cases, no matter what the blood sugar content. In chronic toxæmia, with restlessness and nausea, after every dose of 20 c.c. of 50 per cent. glucose solution, the patient goes to sleep and stops vomiting, and in many of these dangerous disturbances the addition to the glucose of the thyroid residue seems to intensify the beneficial effects of the glucose. This non-coagulable extract, by the way, does not in these cases accelerate the pulse rate.

Apparently, therefore, the thyroid residue makes the glucose more available for assimilation by the nervous system. Hence, in the speaker's opinion, it seems probable that normally the thyroid product helps the nervous system to absorb glucose; that in hyperthyroidism there is some change in the character of the thyroid product which interferes with this absorption; that there may be an excessive quantity of secretion, but the quantity is far less important than the quality; that in the severe and more or less acute toxæmias the quality of the product is so poor as to be entirely incapable of providing the nervous system with sugar; that in the very acute post-operative toxæmias the thyroid product, because of the traumatism to the gland, is for

a time entirely absent and the ensuing restlessness and delirium are evidences not of too much poisonous thyroid product, but of a sudden and complete failure of an already half-starved nervous system to obtain any more sugar. This thyroid residue is admittedly an imperfect substitute for the normal product of the gland, but it is the best now obtainable and it undoubtedly bridges over the interval until the injured gland can at least partially functionate.

Dr. Howard Lilienthal asked if the addition of insulin to the administration of glucose and thyroid residue would be of benefit in these cases of lowered natural production of thyroid secretion.

Dr. John Rogers replied that the use of insulin in these cases may be dangerous. That might seem to be a curious contradiction in these cases of toxæmia in which it is necessary to administer 10 c.c. of glucose every 24 hours to keep the patient alive. But in one case he had tried the experiment of giving 10 units of insulin after the glucose and the patient became delirious, had convulsions and rapid rise of temperature. She pulled through, but if she had had more insulin it was the speaker's belief that she would have died.

The thyroid residue, by the way, is the trade name for an aqueous extract of the thyroid from which all coagulable material has been removed. This non-coagulable liquid is then concentrated so as to contain a definite amount or iodin.

BILATERAL MASSIVE COLLAPSE OF LUNG

Dr. Harold E. Santee remarked that three years ago, Doctor St. John read a paper before this Society on massive collapse of the lung. Since 1908, when Pasteur described this as a true clinical entity, the work of Bradford on gunshot wounds of the thorax, and particularly, the careful reviews of Scott and Churchill last year with the report of cases from Peter Bent Brigham and Massachusetts General Hospitals, together with the previous reports of F. A. C. Scrymger, leave little doubt as to the frequent occurrence of this condition but considerable doubt as to its true significance. Personally he had held a somewhat sceptical attitude in regard to any real significance attached to this condition and had viewed it as an interesting post-operative clinical finding only, but in the light of the autopsy findings here presented, he had changed this opinion and now feels that it may be much more significant than he realized. These cases with two other autopsy cases will be published in full by Doctors Bergamini and Shepard of the Second Surgical Division at Bellevue and he wishes to acknowledge the courtesy of these men and of Dr. Charles Norris of the Medical Examiner's Office in granting him the privilege of presenting these specimens. Only brief outlines will be presented:

Case I.—M. L., age forty-four, died on the operating table on September 24, 1926, just at the end of a hysterectomy and appendectomy. General condition had been good previously and also during the operation up to the time of a complete cessation of respiration. The usual methods of resuscitation plus the pulmotor and adrenalin into the heart were unavailing. Autopsy

showed massive collapse of left lung and collapse of all but one lobe of right lung. No bronchial obstruction was found and the only significant additional finding at post-mortem was a moderate œdema of the larynx.

CASE II.—The gross specimens of this case are presented. The history is surrounded by conflicting statements. From these it is gathered that sudden death occurred in an office, presumably in connection with tonsillectomy, but septic abortion was diagnosed. No odor of anæsthesia was present. Collapse of the lung was massive on both sides with no bronchial obstruction made out. The fresh gross specimens of each case can be held in the cupped hands. No hardening or shrinking fluid has been used on the specimen presented. The microscopic preparations in both cases, as examined by Doctor Symmers, reveal a tissue which it is difficult to recognize as lung, resembling, rather, a solid organ. This appearance is found to be due to complete atelectasis of the pulmonary alveoli, the epithelial cells of which are closely packed together, having lost entirely their normal alveolar arrangement. The individual cells are swollen, certain of them being obviously hydropic, and the cell outlines are rather indistinct. The bronchioles are also collapsed for the most part, many of them being represented merely by circular clumps of cuboidal cells. The capillaries, arterioles and venules, on the other hand, are all uniformly dilated and filled with blood, producing almost an angiomatous appearance in certain areas. This constitutes the most characteristic feature of the histology of the condition.

In the various explanations advanced as to the cause of this condition, paralysis of the muscles of respiration as outlined by Pasteur cannot constitute the whole cause. Mechanical plugging of the bronchus certainly does not explain all cases. The best explanation seems to be that there is some reflex disturbance which causes a constriction in the air passages, probably affecting the small bronchioles, not dependent originally on infection and acting on both lungs to some extent. In this explanation as outlined by Scott, the following possibilities present themselves:

(1) Vasomotor disturbance—dilatation and stasis; (2) bronchiole spasm; (3) swelling of the mucous membrane similar to angioneurotic œdema.

In the specimens presented are factors seeming to support all of these possibilities to some extent, but the true causative mechanism is still a matter of conjecture.

DR. HOWARD LILIENTHAL had noticed during the War that there sometimes occurred after a gunshot wound in the chest what was called traumatic atelectasis, but he believed this was the same thing that happens in massive collapse of the lung. In the unopened chest the alveoli become filled with cells and the blood-vessels become engorged, because actual collapse could not occur. He was glad to hear there was no bronchial obstruction in the cases that came to autopsy. From what Chevalier Jackson said, the speaker had had an idea this condition was caused by the plugging of the bronchial branches because he has reported cases where this plugging was removed and the collapse disappeared. Doctor Lilienthal had always believed it was due to spasm of the lung itself due to some unknown cause. He had suggested to the Surgeon General that experiments be made to find out the cause of this catastrophe, by stimulating the nerve centres. He had talked to Scrymger in Montreal and had asked him what was thought of Jackson's theory and he

said he did not believe it fitted all cases. Here is Doctor Santee's case in which a full post-mortem was made and no obstruction of the bronchi found. This is a very important contribution.

SPONTANEOUS DISLOCATION OF A SUBSTERNAL GOITRE

Dr. William Barclay Parsons presented a woman who was twenty-five years of age in May, 1924, at which time she complained of nervousness, palpitation and goitre. Ten years before admission she began to have a choking sensation with nervousness, palpitation and tremor of her hands. Insomnia appeared shortly. Three years after onset she extended her neck violently and had a feeling that something moved in her neck. Simultaneously the goitre appeared and the choking sensation disappeared never to return. She had a + 50 basal metabolism, tachycardia, nervousness, tremor and insomnia and was operated upon with a good recovery.

He had never heard of this occurrence, namely a spontaneous dislocation of a substernal goitre.

LATE POST-OPERATIVE RECURRENT NERVE PALSY

Doctor Parsons presented a man who was fifty-two years of age in March, 1924. He came to the hospital with a complaint of dyspnœa for ten years which had been gradual in development without cough or palpitation. Dyspnœa was markedly increased with the slightest respiratory infection but there had been no toxic symptoms.

On physical examination he presented curvature of the spine thought to be due to an injury in infancy and a soft boggy swelling just above the right clavicle, the mass apparently passing down into the thorax. On X-ray there was a substernal shadow, a narrowed trachea that was displaced considerably to the left and an apparent downward displacement of the aortic arch. Laryngoscopic examination by Doctor Babcock showed a marked laryngeal displacement to the left without any vocal cord paralysis. Basal metabolism was ±0.

Operation was done under local with the Pemberton technic. A 9 cm. x 5½ cm. mass was removed from the right side. The left lobe was small and soft. He made an excellent recovery and was discharged on the tenth day with distinct improvement in his respiration. His voice was perhaps a little hoarse but not more than many individuals on the tenth post-operative day and there was a distinct vocal quality to it.

He went up to Burke and after about a week, on the seventeenth or eighteenth post-operative day, his voice altered in the afternoon and by night he was almost aphonic. He had no pain and no cough. He returned to New York about a week later and Doctor Babcock on examination this time found a partial paralysis of the right vocal cord. At follow-up at six months his voice had returned, but he was hoarse. Laryngoscopy showed the right cord still paralyzed, the left beginning to over-act. At twelve months his voice was almost as good as before operation and Doctor Babcock noted full compensation by the left cord. At twenty months he returned from abroad, having put on considerable weight, so he began dieting under Doctor Bauman, and from November, 1925, until June, 1926, by great faithfulness, had reduced twenty-five pounds and was in much better shape. At twenty-seven months the result was very satisfactory. He was breathing easily and his voice was, to his mind, as good as before the operation.

It is rather unusual for a recurrent nerve palsy to appear at this interval. In the reported cases there was either a section at operation in which the paralysis came on immediately or else there was a contraction of scar tissue

which did not cause the palsy until several months, usually four to six. I imagine that this man's nerve was caught in the shift of planes which took place and became angulated or constricted earlier than usual. It is hard for me to believe that his nerve was sectioned at operation inasmuch as local was used and he talked very well during and immediately following the operation.

Frazier, of Philadelphia, discussed before this society last year his results in nerve repair in these cases. In view of this man's reasonably good voice it does not seem worth while now to advise this procedure.

ADENOMA OF THE THYROID

Dr. William Barclay Parsons read a paper with the above title, for which see page 107.

Dr. Henry W. Cave recalled having seen a young man who had yawned rather heavily and had immediately experienced the sensation of choking to death and was blue in the face. He had dislodged a large sized substernal adenoma of the isthmus. He was taken to the hospital and operated on under local anæsthesia, and it was found that he had an enlarged thymus gland—a status lymphaticus. Three months later there was acute œdema of the thymus which may have had something to do with the pushing up of the adenoma of the isthmus.

Dr. Charles G. Heyd said that the hyperthyroidism of adenoma behaves differently clinically from the hyperthyroidism of Graves' disease. There is first of all the difference in the age grouping of the two conditions, and secondly, in the clinical manifestation. He believed that the hyperthyroidism of Graves' disease was really a dysfunction in that there was some inherent change in the chemistry of the secretion of the thyroid in Graves' disease that was essentially different from the secretion of the thyroid in adenoma. Most of the patients with an adenoma that develop hyperthyroidism have had a tumor of the thyroid for long periods of time, so long in fact that they had accepted the goitre as a matter of fact and presented themselves in the dispensary, or at consultation, for a heart complaint, or a tachycardia, or other symptoms of hyperthyroidism.

In regard to iodine medication Doctor Heyd was impressed with the fact that a great deal of harm was being done by the wholesale administration of iodine. His own cases of adenoma that had been treated with iodine had been more dangerous from an operative point of view than an ordinary uncomplicated case of Graves' disease. Apparently, iodine in an adenomatous goitre, was fraught with two dangers, first, the danger of converting a non-toxic adenoma into an adenoma with hyperthyroidism, and secondly, if given in a hyperthyroidism of adenoma it was very apt to produce a "fixed" hyperthyroidism. In other words, a hyperthyroidism that did not respond to rest and medical therapy or the measures that were used as a preliminary to operative intervention. The cases of fixed or iodine hyperthyroidism offered a greater operative risk than did the simple cases of hyperthyroidism that have not been given iodine. If he assumed the correctness of Plummer's

theory of iodine in Graves' disease it was indicated only for a short period of time for the purpose of lessening an operative risk and in his opinion was about equivalent to a bipolar ligation.

Doctor Heyd wished to draw attention to the routine post-operative treatment of goitre cases that consisted in the administration by rectum of 500 c.c. of a 10 per cent. glucose solution with 4 c.c. of Lugol's solution every eight hours. Most of the cases of hyperthyroidism that had been operated upon by him were done under rectal anæsthesia with the patient going to sleep in her own bedroom and avoiding thereby all of the exciting causes incident to a trip to the operating room. The surgery of adenoma of the thyroid was, in the opinion of Doctor Heyd, different than the surgery of Graves' disease. If Graves' disease represented a hyperplasia then the operative indication was for a removal of a major portion of the thyroid in order to diminish the secreting units of the thyroid by just the amount represented by the tissue removed. On the other hand, the surgical indication in an adenoma was the removal of a tumor growing within an apparently normal thyroid gland. That this condition seemed to be correct was borne out by the fact that a case with an adenoma with hyperthyroidism usually recovered much more quickly after surgical intervention than did the patient with the hyperthyroidism of Graves' disease. In other words, removal of the offending adenoma of the thyroid brought about a more prompt and a more complete cure or relief from symptoms than did a subtotal resection of the thyroid in the cases of hyperthyroidism of Graves' disease where the recovery was more delayed.

Doctor Parsons, in closing the discussion, said that he agreed with Doctor Heyd in regard to the distinction between the hyperthyroidism of adenoma and that of Graves' disease, but it is hard to prove. In reference to iodine, although he felt that some of these cases were stimulated to activity by the injudicious use of iodine, one must consider this point from the time standpoint in that these patients have carried a goitre for a long time. Something happens which makes them go to a doctor. This may be the beginning of their hyperthyroidism. He gives them iodine and toxic symptoms develop. Is that coincidence, and has the time of the iodine therapy overlapped the development of the hyperthyroid symptoms? If that is so, the iodine therapy was of no importance in the induction of hyperthyroidism, but it seems to happen too often to be a mere coincidence.

Stated Meeting Held January 12, 1927

The President, Dr. Walton Martin, in the Chair

PEDICLE GRAFT FOR OS CALCIS COVERING

Dr. Ralph Colp presented a man, age thirty-five years, who was admitted to the Surgical Service of the Beekman Street Hospital, November 8, 1925. On the day of admission, when attempting to stop an elevator, he missed the rope and inadvertently caught his right foot between the floor of a moving elevator and the floor of the building, crushing the heel.

Examination disclosed a transverse lacerated wound of the right heel down to the tendon Achilles, really a partial avulsion of the heel. The Röntgen examination of the right foot showed an incomplete fracture of the os calcis without displacement. Under anæsthesia the wound was thoroughly irrigated with saline solution for about fifteen minutes and then cleansed with alcohol and ether. After thorough débridement, the laceration was sutured with silkworm gut and silk, and the wound was dressed with vaseline gauze. Dressing a few days after operation revealed that the wound was not infected, but that the lower flap of skin covering the heel; that is an area of about 2 inches x 3 inches, had become black and gangrenous. Within the one week, the necrosis of the heel had progressed so that a circular area about 5 inches in diameter was involved. The underlying granulations, upon removing the slough, however, were fairly clean and in the centre, bone was exposed.

Four weeks after the initial trauma, the wound surface which measured about five by four inches, appeared clean, the granulations red and exuberant, and conditions seemed satisfactory for a pedicle graft.

December 10, 1925, a pedicle graft was transferred to the right heel, employing the following technic: The area of the right heel was thoroughly scrubbed with green soap and water, followed by alcohol and ether, and since this procedure caused bleeding, the granulating area was covered with hot towels and pressure applied by hand of an assistant. A full thickness pedicle graft was cut in the region of the left thigh measuring about 4 inches in length, and about 5 inches in width. The pedicle ran parallel to Poupart's ligament. The thickness of the graft included all tissues between skin and the deep fascia of the thigh. The denuded area which was caused by raising the skin flap was thoroughly covered with Thiersch grafts which had been removed from the anterior aspect of the right thigh. These grafts were held in place with paraffined gauze reinforced by moist gauze dressings. A sheet of rubber dam completely encased this dressing. This dam prevented secretions from the thigh reaching the area of the heel, and the secretions from the heel seeping into the dressings of the thigh. The right heel was then brought up to the anterior aspect of the left thigh where it rested on the rubber dam, and the free edge of the graft was sutured to the periphery of the wound of the heel with interrupted silkworm gut sutures for about one-half of the extent. In order to permit secretions to escape through the graft, it was perforated in several places with a Dakin-tube punch. The parts were then rendered absolutely immobile by a plaster spica, which was applied. completely encasing the lower half of the body. The graft itself was covered with paraffined gauze and a sterile towel. Twelve days later the graft, which was completely viable, was incised at its base for a distance of about one inch on each side. There was free active bleeding from the distal part of the graft. The next day, under local anæsthesia, the flap was entirely divided and the free end sutured to the heel with interrupted silkworm gut. The plaster case was then removed and the area of the left thigh was dressed for the first time. It was found that all the Thiersch grafts had taken.

The patient was discharged January 5, 1926, with the heel completely cicatrized. He was warned because of lack of sensation in the graft to protect the skin from the shoe by means of a layer of rubber sponge. Wound remained completely healed until December 1, 1926, when because of the holiday rush, patient was forced to walk around most of the day and neglected to protect his heel with a rubber sponge, resulting in a small ulcer which has since healed.

Doctor Colp also presented a youth of seventeen years, who was admitted

to the Beekman Street Hospital, March 26, 1926. His history dates back to fourteen months before admission, when his right heel was crushed between the street pavement and the platform of a moving elevator. The wound, which involved practically the entire heel, never cicatrized completely, but left an ulcer about the size of a silver half dollar which did not heal in spite of the fact that it was dressed three times a week for over a year. When admitted to hospital he presented a sluggish ulcer about the size of a fifty-cent piece, lying practically on the posterior medial aspect of the right os calcis. The region of the right heel was replaced by dense scar tissue firmly adherent to the underlying os calcis. There was a deformity of the foot with a contracture of the flexors of the leg resulting in an apparent talipes equinus, although when patient walked, the gait was almost normal. The Röntgen examination of the foot showed an old complete fracture of the os calcis about midway through its body. This was healed with the posterior fragment turned markedly to the inner side, and that it was comminuted is indicated by a hole through the outer side of the bone. There was a rather large deforming callus present.

Inasmuch as the ulcer was placed almost directly upon the os calcis and surrounded by dense scar tissue, a pedicle graft seemed to be the only procedure which might effect a cure. Before this could be done, a bed of healthy granulation tissue to receive the graft had to be prepared. April 3, 1926, the ulcer was thoroughly curetted and a small part of the scar tissue about the os calcis was excised and about a dozen holes were drilled into the uncovered os calcis with the hope that granulations might spring up from the medulla and thus cover the bone. The leg and foot were placed in a plaster case in dorsal flexion. Ten days later the wound was clean and granulations were flourishing and beginning to cover in the bone. There was still a great amount of scar tissue surrounding the area of granulations. It took three subsequent operations before all the scar tissue had been excised and drill holes had been bored in the bone which was uncovered by the removal of the cicatrix. An X-ray picture of the heel did not disclose any infective osteomyelitis. About six weeks after his admission, the region of the heel as represented by the posterior portion of the os calcis, was covered with bright red granulated tissue and the conditions seemed favorable for a pedicle graft. Accordingly, on May 17, 1926, a pedicle graft was applied to the right heel for the large defect which involved the posterior portion of the heel, extending from its inferior margin up on to the region of the tendon Achilles for the extent of 5 inches. The transverse diameter of this defect was about 3 inches. The technic employed was similar to Case I.

Six days post-operative the graft was in excellent condition and the pedicle was partially divided one inch on each side. Good active bleeding occurred from the distal side.

Eleven days after the primary operation, the pedicle was completely divided. The area which had been covered with Thiersch grafts was dressed; all had taken. Three days later, the free end of the pedicle graft was sutured into place with interrupted silkworm gut sutures. Following this there was some sloughing of the graft, the granulating area resulting was covered with five small pinch grafts. The patient was discharged July 3, 1926, and since then the affected area has remained completely healed with an excellent functional result.

These cases were shown not only to illustrate the well-known technic of a pedicle graft and to show the practicability of covering the os calcis with

granulations derived from its own medulla, but to emphasize the importance of pedicle grafts in this region.

The heel because of its anatomical location is made to stand the stress and strain of daily use, large scars, the results of trauma to this region are naturally poorly tolerated, and the resulting chronic ulcers produce total disability in the majority of cases. In wounds of the heel with a loss of substance a full thickness pedicle graft with an adequate supply of subcutaneous fat appears to be the ideal procedure. It should be remembered, however, that sensation is slow to return to the graft and that the heel should be adequately protected from the unsuspected irritation of the shoe until such time as the sensation returns and the skin has adapted itself to its new environment by the development of callus.

TRAUMATIC RUPTURE OF POLYCYSTIC KIDNEY

Dr. James N. Worcester presented a man, forty-eight years old, who was admitted November 3, 1926, to the Beekman Street Hospital. Just before admission he had been run into by a truck, being struck in left flank. Immediate pain in right flank and right side of abdomen. When admitted he presented marked tenderness and rigidity in the right flank and right abdomen. Slight spasm left flank. Quite marked shock. He showed signs of internal hemorrhage. Catheterization produced pure blood. A rupture of the right kidney was diagnosed and an immediate operation was performed. The kidney was exposed by an oblique incision; on opening the capsule a large amount of blood escaped. On inspecting the kidney it was seen that an abnormal kidney was present. It was very large and studded with cysts and in many places was adherent to the capsule. The pelvis of the kidney was completely torn across and the kidney almost split in half. An attempt was made to tie the renal vessels, doubtful if this was successful. On account of profuse oozing, the cavity was packed with gauze.

Post-operative.—Immediately following operation condition not very good; however, he responded to hypodermoclysis and proctoclysis. Following which convalescence was remarkably smooth. Packing removed on sixth day without hemorrhage and tube inserted. This was gradually shortened. Wound completely healed on discharge. For several days post-operative ran blood in urine and some white cells. Quantity always satisfactory. Specific gravity always low, 1008–1012.

December 1, 1926 Blood chemistry as follows: 20.6 mg. urea nitrogen in 100 c.c.; 2.1 mg. creatinine nitrogen in 100 c.c. Phthalein test: Appearance in eighteen minutes—25 per cent. first hour, 20 per cent. second hour; total, 45. Urine amber, acid, 1010 clear, no casts. Present condition is satisfactory. The left kidney can be felt extending almost down to the spine of ileum.

MEDIASTINAL SARCOMA—TREATED BY COLEY'S FLUID

Dr. Howard Lilienthal presented a female aged four years, who was brought to the Clinic of the Hospital for Joint Diseases, March 21, 1924. The child had been apparently normal; had weighed nine pounds at birth; nursed for ten months; stood up at eight months; talked at ten months and walked at sixteen months; teeth at eleven to twelve months. The child had gained slowly in weight, although there had been frequent green diarrhœal stools but without vomiting.

At the age of five months the mother noted that its breathing was distinctly labored but the doctor could find nothing wrong with the lungs. For

two months there was respiratory disturbance but without cyanosis or other sign of deficient aeration. The child's color was pale and the heart's action often rapid. The mother stated that she noted a lump "on the right shoulder blade" at the age of about eight months and she took the infant to the Babies' Hospital, where an X-ray study was made. This was reported as showing: "A large roughly quadrilateral dense shadow in the lower part of the right chest extending over the heart; also into the left chest and down over liver shadow. Right chest above this appears free from lung tissue (!). The right diaphragm appears adherent to the mass. The right chest is smaller than the left. The right bronchus not seen. Probably congenital atelectasis." This report was kindly furnished by Dr. Kenneth D. Nichol, Resident Physician of the hospital on December 16, 1926.

The mother refused to leave the baby but took it to the Lebanon Hospital, where the X-ray report made on April 30, 1923, was as follows: "Fluoroscopic examination of chest of Phyllis H., shows a dense shadow, homogeneous in character, sharply circumscribed, ascending apparently from the lower mediastinum and projecting to the right. The mass is the size of a small orange; its left border projects slightly to the left of the median line and is overshadowed by the heart. The appearance is that either of a cyst or neoplasm ascending from the mediastinum. From its shape and age of the patient we are inclined to believe that it is very likely a cyst."

A puncture was made at the Lebanon Hospital which produced sterile bloody non-coagulating fluid. The Wassermann test and the usual blood and urine examinations failed to show positive indications of disease. Operation was suggested at this time, but the mother refused, and took the child home.

At twenty months of age tonsillitis developed and the patient was taken to Beth David Hospital, where röntgenological examination resulted in corroboration of the former findings.

March 16, 1924, when twenty-one months old, the child suddenly stopped walking because of weakness of the right lower extremity. This became rapidly progressive. At first she was able to stand, but the right foot turned out and she fell on attempting to walk.

When seen by Doctor Pollak at the Hospital for Joint Diseases, it was noted that the patient was a bright, well-nourished child without fever or pain. There was great weakness of the lower extremities, particularly of the right. There was total inability to stand because of paresis of both legs. There was a mass between the right shoulder blade and the spine with dulness upon percussion; breath sounds were exaggerated anteriorly. The blood examination revealed hæmoglobin, 42 per cent.; red blood cells, 3,200,000; leucocytes, 14,000; polymorphonuclears, 42; small lymphocytes, 38; large lymphocytes, 13; transitionals, 1; eosinophiles, 3. Urine examination negative. X-ray studies by Dr. H. B. Phillips on April 14, 1924, resulted in the following report: "Cyst lower right chest. Pressure erosions, spine and rib—and œsophagus is displaced anteriorly." On May 16, Doctor Phillips made another röntgenological study of this case. The final röntgenogram, which is shown herewith (Fig. 1), was by Doctor Jaches.

The child was first seen by Doctor Lilienthal, April 12, 1924, one month after she had stopped walking because of the constant weakness of the right hamstring muscles. At that time, both legs were flaccid and obviously paralyzed. There was a protruding subcutaneous mass covered with normal skin between the right scapula and the spine. It was firmly elastic. X-ray examination showed a large mass in the right chest extending across the

median line into the left chest above the heart. (Fig. 1.) The opacity of the right side occupied the lower two-thirds of the chest and extended to the left above the heart where there was a large shadow. There appeared to be partial erosion of the adjacent ribs posteriorly and also some erosion of the bodies of the neighboring vertebræ. The child's condition was so good, however, that it was hard to think of the case as one of malignancy.

The patient having entered Mt. Sinai Hospital, on April 15, Doctor Lilienthal operated, using at first local and then general anæsthesia. He resected about one and one-half inches of a rib subperiosteally over the tumor. He then aspirated with a coarse needle a minute quantity of thick, bloody fluid. The posterior mediastinum was then opened and the capsule of the tumor exposed. This was incised so as to admit the index finger. The wall was tense and the tumor rudely spherical. A large part of the contents was removed with the finger. It consisted of soft grayish-red neoplastic tissue. The total amount removed was a little greater than the bulk of a golf ball and the oozing cavity was packed with iodoformized gauze. This gauze was allowed to protrude at the ends of the wound and the skin was closed with silkworm gut sutures. The pleura was not entered. The tumor was examined at the laboratory by Dr. F. S. Mandlebaum with the following conclusions:

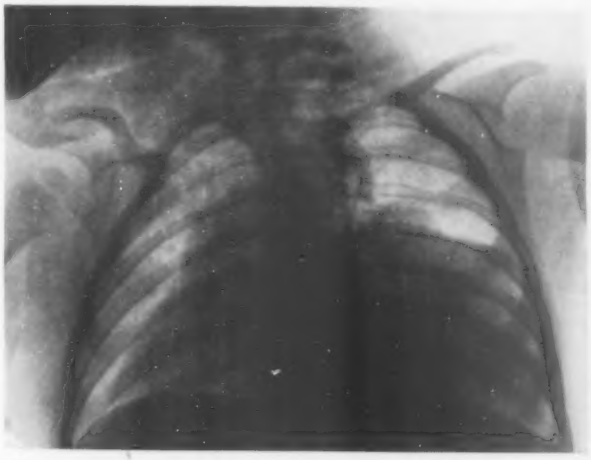

FIG. 1.—Röntgenogram showing condition on March 20, 1923. Before operation.

"Microscopical examination of the specimen removed from the mediastinum of Phyllis H., shows a tumor composed of rather large round cells of uniform size with a vesicular cytoplasm. A large number of thin-walled blood-vessels are present throughout the tumor tissue and the round cells appear to have a close relationship to the walls of these vessels. For this reason one is justified in making a diagnosis of angiosarcoma rather than lymphosarcoma which ordinarily would be made in a tumor presenting this type of cells. The derivation of the tumor cannot be determined from the material submitted."

The wound healed nicely and it was advised that treatment with Coley's mixed toxins should be instituted. This was carried out by Doctor Pollak beginning April 25 with one twenty-fifth of a minim. This was followed by severe reaction. For eleven days the injections were continued in increasing doses up to 4½ minims. After this the treatment was refused by the mother on account of the severity of the reaction phenomena.

May 6, 1924, only three weeks after the operation, the child began to walk, the relief apparently having been due to decompression. However, the improvement continued and a series of X-ray pictures by Dr. H. B. Phillips showed gradual disappearance of the tumor mass until at the last observation, October 13, 1926, an X-ray picture by Doctor Jaches revealed what

he considered a normal chest. The child has remained apparently well. (See Figs. 1 to 3.)

In order to get a possible sidelight on the histological picture, Dr. Louis Gross, Director of the Laboratory of Mt. Sinai, was consulted, who gave an opinion which coincided with that of Doctor Mandlebaum, except that his terminology was "hemangio-endothelioma."

Dr. James Ewing was good enough to examine the same slide and dictated the following: "Malignant cellular tumor of embryonal type composed of many blood sinuses lined by two or more rows of tumor cells. Very delicate stroma." It was his opinion that we were dealing with an extremely malignant form of tumor.

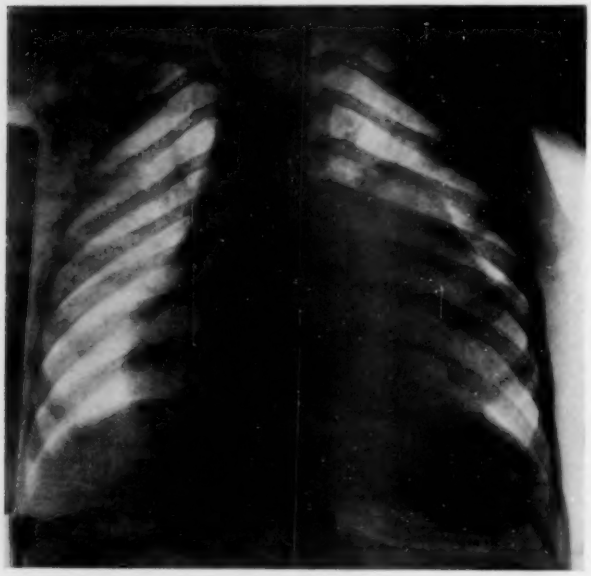

FIG. 2.—Large right shadow still present, though smaller, 17 months after Fig. 1. Tumor of left side practically gone.

The child is presented, walking and with nothing to show for her experience with malignancy except the resulting scar in her back. It is now almost three years since the operation.

Comment.—It will be noted that this child received no treatment of her malignant condition except the administration of the toxins and although the injections were few in number, yet the reactions were excessive. This is not the first time that the reporter had noted a continuance of the recession in cases of malignancy after this treatment had been discontinued.

DOCTOR LILIENTHAL added that while he presented this case as an isolated one, yet his experience with this form of therapy in a number of other instances had been so favorable that he would strongly recommend its use in inoperable sarcoma and also as a prophylactic post-operative treatment after the surgical removal of operable tumors as well. With the latter object in view, it is not necessary to give doses large enough to produce more than moderate reactions. It should also be noted that in nearly all cases of inoperable malignant growths there are metastases whether demonstrable or not and any treatment which is directed merely to the site of the primary or principal growth should not be expected to affect the distant secondary tumors even though they be of microscopic size.

The action of the toxins is one which affects the entire organism. If there is any selective property in this form of therapy it is manifestly to be preferred to measures which are efficient only at the site of application. In this respect the toxins may be compared to the colloidal lead treatment of

neoplasms which is being tested at present and with encouraging success by its sponsor, Blair Bell, of Liverpool. In the lead treatment, however, there are at least two disadvantages—first, that of lethal plumbism, and second, the danger of the perforation of hollow viscera which may be the seat of neoplastic disease. When properly administered, Coley's toxins are not directly dangerous to life. There are, of course, reactions, often very severe, but these can be controlled by proper dosage and when all febrile signs have disappeared, which they usually do in a few hours, there is no further danger from the individual dose. Coley has reported numerous cures which have been effected by the toxins and the reporter himself had been fortunate in observing a number of instances in which there has been apparently permanent disappearance of the disease. Some of these will be reported in a forthcoming book which is being prepared by Doctor Coley.

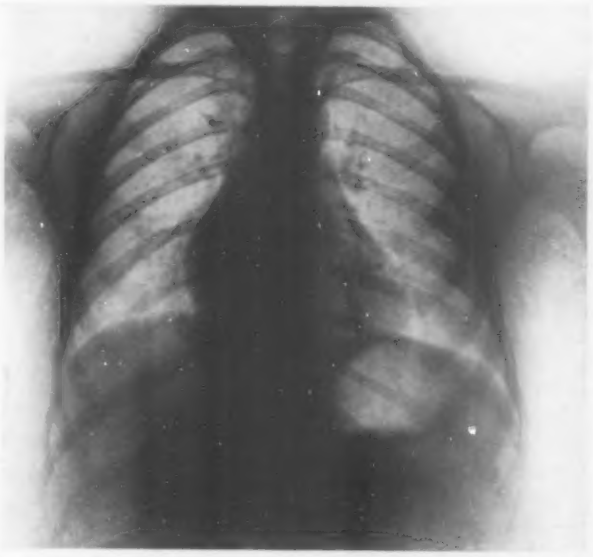

FIG. 3.—The same patient 2½ years after treatment by toxins. Doctor Jaches reports this chest as normal, not counting, of course, the slight changes which may be observed in the ribs on the right side.

Coley believes that about 10 per cent. of successes may be expected. It was the speaker's impression that this estimate is not exaggerated. At any rate, he was convinced that there is nothing which holds out similar hope in the treatment of inoperable malignancy.

DR. WILLIAM B. COLEY stated that he had observed seventeen cases of sarcoma of the spine or sacrum, in all of which the disease had reached the inoperable stage at the time of his first observation. In addition, two other cases of inoperable sarcoma of the spine had been treated with the toxins by other surgeons under his direction, making a total of nineteen cases in all. This series included ten females and nine males. The locality of the tumor was as follows: Two cervical, seven dorsal, seven lumbar and three sacral. A microscopical examination was made in all but two of the cases, and the following classification given: Nine giant-cell, four spindle-cell, two mixed-cell, and two round-cell. The age of the patients ranged from ten to sixty years. Nine, or 47 per cent., of the nineteen cases remained alive and well from three to twenty-four years; one patient died of a recurrence at the end of six years. In the nine cases in which recovery took place, the toxins alone were used.

DOCTOR COLEY gave a brief history of some of the most noteworthy of these cases as follows:

CASE I.—D. G., male, aged twenty years, was always in good health until the latter part of 1901, when he developed a swelling in the mid-dorsal region. This grew rapidly and partial paralysis of the lower extremities developed a short time later. He was sent to the Montefiore Home for Incurables in February, 1902. Examination at this time showed a very large tumor occupying at least five or six of the dorsal vertebræ. The patient had complete paralysis of the bladder, rectum, and lower extremities; and had lost fifty pounds in weight. The condition seemed to be absolutely hopeless. He was put upon the mixed toxins of erysipelas and Bacillus prodigiosus, which treatment was kept up for four months. The improvement in his condition was immediate and marked; in a few months he was able to walk about with a plaster jacket. His recovery was complete; and a few years later, he married and now has two children. Physical examination in March, 1924, showed the patient to be in excellent condition and he is still in good health, twenty-four years since the treatment was begun.

Dr. Harlow Brooks, Professor of Pathology, Bellevue Hospital, examined a section of the tumor microscopically, and pronounced it a round-cell sarcoma. Numerous atypical giant-cells were present; but whether or not the tumor would ever metastasize, it was impossible to say. At any rate, there was no question but that it was highly malignant locally, and of very rapid growth, and, undoubtedly, would have killed the patient in a few months had it not been controlled by treatment.

CASE II.—Baby W., male, aged two years and nine months, was first seen in March, 1911. In February, 1911, the father, while holding the child in his arms, was thrown out of a wagon. Nothing was noticed at the time, but the spine of the child was undoubtedly injured. One month later he began to get weak, and in the latter part of March a swelling was noticed about two inches above the upper border of the clavicle on the right side. This increased rapidly in size; and the patient continued to lose power of his muscles. The condition was regarded as infantile paralysis. There was marked muscular tremor of both hands, and almost entire loss of power in the arms; the patient could not move himself at all, neither was it possible for him to hold up his head. An exploratory operation was done by Dr. Stuart McGuire, of Richmond, Virginia, which revealed the presence of a well-defined tumor springing from the laminæ and transverse processes of the cervical vertebræ. A section was examined microscopically, and pronounced fibro-sarcoma.

The toxin treatment was begun in July, 1911, and kept up for one year. The child's weight steadily increased during the course of treatment; he regained considerable power so that he was able to move his hands and legs, and could hold up his head. Doctor Ewing examined a section of the tumor microscopically in March, 1922, and stated that while he was unable to offer a positive and exact diagnosis, he was certain that it was a malignant tumor which might well be called sarcoma. He was inclined to regard it either as an endothelioma secondary to the cerebral growth or possibly a neurocytoma derived from misplaced nerve tissue in the cranium.

In May, 1921, Dr. Harvey Cushing performed a laminectomy, and found what he considered a very extraordinary tumor plastered all along the spine, though entirely extra-dural. While Doctor Wolbach, pathologist, at first called it a ganglio-neuroma, on further examination he pronounced it a neuroblastoma of an undifferentiated type.

There can be little doubt that the tumor found by Doctor Cushing was an entirely different type from the original tumor which clinically and microscopically was unquestionably a highly malignant tumor. That this extensive inoperable tumor completely disappeared under no other treatment than the mixed toxins, and the patient remained well for more than fifteen years, is, certainly, a gratifying result.

CASE III.—H. B. H., male, aged thirty-eight years. In February, 1895, the patient began to lose flesh and strength. Shortly afterward he felt pain in the lower part of the spine over the sacrum and extending down the legs. It was more marked on the right side. On May 2, 1895, he was admitted to St. Luke's Hospital, where he was examined by Dr. F. P. Kinnicut and the other members of the staff. In the opinion of all, the patient was suffering from an inoperable sarcoma of the sacrum and pelvis. He had lost forty-one pounds in weight and could walk only with the greatest difficulty.

May 10, 1895, a brief trial of toxin treatment was begun. The injections were made directly into the buttocks. At the end of one week, the pain had almost entirely subsided; and the lameness was much improved. At the end of seven weeks, the patient had gained twenty-eight pounds in weight and appeared in excellent condition. He remained well and free from recurrence for seventeen years, when, in an accident, he received an injury to his skull which resulted in his death.

CASE IV.—The following case of recovery from an inoperable osteosarcoma of the vertebræ was reported by Doctors Miketta and Oliver, of Cincinnati, in the *Lancet Clinic*, May, 1910. The history in brief was as follows: A young girl, aged sixteen years, first noticed pain in the upper cervical region in 1907. A small lump appeared in the region of the posterior cervical glands in 1908; and in 1909 the symptoms became more pronounced. In August, 1909, she developed paralysis; there were paralegic symptoms from the neck downward. Röntgen-ray examination by Doctor Oliver showed an osteosarcoma of the second cervical vertebræ, which diagnosis was confirmed by neurological examination.

The toxin treatment was begun in October, 1909, and continued until February, 1910, a total of 52 injections being given. Marked reactions and severe chills (sometimes lasting forty-five minutes) followed the injections. The temperature, however, rarely rose above 99 to 101°; and only on one occasion did it reach 103° F. An injection was given every other day, but after a severe reaction was produced, one only every third day was given. Only slight improvement in the condition was noticed at first, but at the end of two months, the improvement was more rapid. Motion and power returned in the head and arm first, and then in the lower extremities. In April, 1910, the patient was able to perform every movement. She made a complete recovery and was still well when last traced, seventeen years later.

CASE V.—Another case is that of a giant-cell sarcoma of the sacrum involving the coccyx. In May, 1912, an operation was performed at the Mayo Clinic consisting of removal of two lower segments of sacrum as well as the coccyx. Administration of the toxins was begun immediately after operation. The treatment was kept up for about two months in doses sufficiently large to produce severe reactions. The patient was well when last traced, eleven years later.

CASE VI.—In August, 1918, Doctor Coley, in consultation, saw a female adult at Mt. Sinai Hospital, in which a diagnosis of sarcoma of the lower dorsal spine had been made. Röntgen-ray examination showed a tumor of the middorsal vertebræ, and there was paraplegia of the lower extremities.

The toxin treatment had already been started, and, on Doctor Coley's advice it was continued. Marked and steady improvement in the condition was noted, until the patient had completely recovered the use of her limbs. When last traced, two and one-half years later, she was in excellent health and able to do her own house work.

CASE VII.—Dr. Torr Wagner Harmer, of Boston (*The Boston Medical and Surgical Journal*, March, 1915), in a series of cases of inoperable sarcoma treated with the toxins, reported the following: A boy, aged sixteen years, had fallen striking on his spine. Pain and tenderness persisted in this region, followed by numbness over the external and anterior surface of the righ thigh, with absolute loss of sensation. The patient was admitted to the Massachusetts General Hospital three months later, at which time he walked with slight instability, complaining of weakness of right knee, and intense pain in lower dorsal region of spine. He was operated upon by Doctor Porter, who removed the transverse process, curetted the bone, and trimmed the capsule as much as possible. Doctor Hartwell examined it microscopically and pronounced it a "richly cellular tumor composed of spindle cells with numerous giant cells. The cells are very atypical and their richness varies in different areas of the tumor, some places being quite fibrous. There are scattered necrotic areas in the tumor tissue." Treatment with the mixed toxins was started at once and continued for seven months."

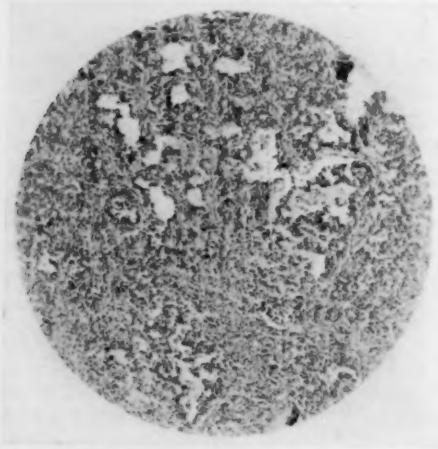

FIG. 4.—Photomicrograph (by Roy M. Allen, magnification 75 x) of section of tumor from the Pathological Laboratory of Mt. Sinai Hospital. See report of Pathologist.

Injections were given every other day and then twice a week until a total of 62 had been given; the maximum dose was 18 minims. During the early course of treatment, the injections were made systematically into the belly, legs, and arms, and later into the recurrence which rapidly developed and grew until it measured 5½ inches in length, 3½ inches in breadth and 2 inches in elevation. There was paresis of the right leg. The treatment was kept up in spite of the violent reactions which occurred. The tumor began to slough in several areas and in January, 1913, there was no evidence of any mass nor any disturbance of sensation of limbs. The patient was in excellent health when last traced ten years later.

DOCTOR COLEY believed the result obtained in Doctor Lilienthal's case showed the importance of not abandoning all hope even in the seemingly quite desperate cases. While, he states, it is too early to pronounce this patient definitely cured, the fact that she has remained in excellent health for more than three years furnishes good ground for believing that the result will be a permanent one. Cases treated with the mixed toxins differ from those treated by radiation or even surgery, in that, in the former group once the tumor has entirely disappeared a recurrence seldom takes place and the patient usually remains permanently well. In proof of this he cited the

results found in a recent follow-up of 93 cases of inoperable sarcoma treated with the toxins alone, reported by him before the Johns Hopkins Medical Society in 1896, as follows: 16 patients have remained alive and well from eight to thirty-three years later, and twelve from ten to thirty-three years. Doctor Coley stated that when we consider how very few cases of malignant tumor, either sarcoma or carcinoma, there are on record in which the patients have been found to be alive and well ten years after treatment, the results obtained in the series just quoted become extremely important. Doctor Coley called attention to the report of Doctor Gibson in the ANNALS OF SURGERY for August, 1926, covering 437 cases of all types of malignant disease operated upon at the New York Hospital, in which only 64 patients were found to be living and without recurrence, and only 13 had reached the five-year limit.

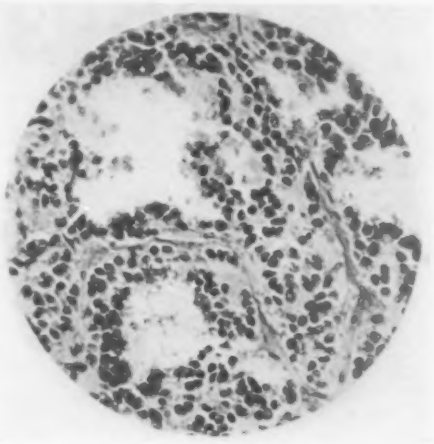

FIG. 5.—Same specimen. Magnification 350 x.

DOCTOR COLEY stated that Doctor Lilienthal was not only one of the first to be convinced of the value of the toxins in inoperable tumors, but that he was among the first to recognize their value as a prophylactic after operation; and that he had employed this method as a routine measure for twenty years or more. The method, according to Doctor Coley, is based on a clinical observation of a considerable number of cases of all varieties of malignant tumors which have disappeared or shown improvement during an attack of intercurrent erysipelas or as a result of inoculation of living cultures of the streptococcus of erysipelas. In a paper published in the *American Journal of the Medical Sciences* in May, 1893, Doctor Coley collected from literature, 38 cases of malignant disease, in 23 of which an attack of erysipelas had occurred accidentally, and in 15 of which it was the result of inoculation. This series included 17 cases of carcinoma, 17 cases of sarcoma, and 4 cases of either sarcoma or carcinoma. In the group of carcinoma, 3 were permanently cured, 1 was well five years later, and in 10 cases decided improvement was noted. In the group of sarcoma, 7 patients were alive and free from recurrence from one to seven years after the attack of erysipelas. Eschweiller, in a monograph published some years later, made a very complete study of accidental erysipelas associated with malignant tumors, collecting 69 cases from literature. This series included 27 sarcoma, 38 carcinoma, and 4 cases in which the type of disease was undetermined. Of the sarcoma, there were 9 apparent cures, and 4 cases in which the patient had remained well from two to eight years; of the carcinoma, there was complete disappearance of the tumor in 6 cases, in three of which there was a later recur-

rence. In six cases the patient died of the attack of erysipelas. Since the publication of that paper, Doctor Coley has collected quite a number of additional cases.

Doctor Coley stated that in the last ten years, so much attention had been given to radium and Röntgen-ray treatment of malignant tumors that the value of the method of treatment with the mixed toxins of erysipelas and bacillus prodigiosus had been lost sight of, or relegated to the background. He stated that if treatment by radiation could produce better results than those obtained by the older method then there was no occasion whatever for reviving the latter. He wished to call attention to the fact, however, that a careful comparative study of the end results obtained by the use of the various methods showed that neither radium nor Röntgen-ray had been able to effect anything like the number of actual cures in inoperable sarcoma that have been obtained by the use of the toxins.

Dr. Alfred W. Pollak said that in the case of the child who had been presented the intensity of the reactions following the use of Coley's fluid was very great. But from the very first injection the healing of the operation wound was extraordinary, and after the third injection the progress of healing of the wound was even ten-fold quicker than before, and after the adverse reaction was over the child seemed to be better generally. The hæmoglobin count was 42 per cent. at the time of operation and after the eleventh injection it went down to almost 30 per cent. Because of the violent reactions the mother requested that the injections be stopped, at least temporarily and Doctor Lilienthal thought this might be done without ill effects and a transfusion be given. While the giving of a transfusion was being considered, the improvement in the patient became so marked that matters were allowed to stand as they were and in a comparatively short time the child was up and around. Last summer, after the tumor had disappeared, very severe whooping-cough developed, there being as many as fourteen violent paroxysms in one afternoon. During the course of this whooping-cough a röntgenogram was taken of the lungs to see if they were affected, but the picture was negative. A severe attack of measles and then of bronchitis followed the whooping-cough, but cleared up without any ill effects.

Doctor Lilienthal, in closing the discussion, affirmed what Doctor Coley said, in that he is a consistent user of the toxin, but not all of his patients had survived. Nevertheless he considered that Doctor Coley's claims were not exaggerated, for he had had many patients cured who had remained well for a long period of time. Doctor Lilienthal had only one thing more to say and that was a suggestion. Doctor Coley had spoken of the scientific basis for the treatment. It was really empirical in the beginning, founded on the supposed action of erysipelas on sarcoma, which has been known for many, many years. Sir James Paget brought that out at least fifty years ago. (Clinical Lectures and Essays.) Much of the good work

done in medicine has started as empirical, been sustained by theory and then by anatomy and stood at last on a firm scientific basis. It seemed to the speaker that in this toxin treatment one might find a scientific basis. According to the theory advanced by Gye, it probably takes more than a germ to produce malignancy, so it takes more than a germ to destroy it; Gye believes that it takes a certain chemical substance in the blood together with the germ to produce malignancy. This gives us a suggestion as to why foreign proteins act in producing a cure; they probably produce some change in the physiological chemistry in the body which destroys the medium for the germ of malignancy.

TUBERCULOSIS OF THE BREAST

Dr. Burton J. Lee presented a woman, forty-five years of age, who was admitted to the Memorial Hospital, May 9, 1924. Four months prior to admission, she first noticed a small lump just above the nipple of the left breast. The mass had seemed to remain stationary in size.

Fig. 1.—Tuberculosis of breast. (Chest plate.)

When admitted examination of the left breast revealed, beneath the areola, a small mass about one centimetre in diameter, which felt cystic. The skin overlying this mass was slightly reddened, the mass itself was somewhat fixed to the overlying skin and through one small sinus opening, a slight amount of yellow serous discharge was exuding. Surrounding this mass, the breast, itself, seemed slightly indurated for a distance of about two centimetres. The nipple was not retracted and there was no discharge from the nipple. Axillary lymph-nodes were palpated in both axillæ, there being two nodes palpable on each side. These were moderately soft in consistence and not tender. Examination of the right breast showed a slight degree of indefinite nodulation throughout the breast.

Röntgenographic examination of the chest made by Doctor Herendeen showed some shadows at the apices, suggesting old, healed tuberculosis lesions.

May 10, 1924, the mass was excised through normal breast tissue, going well wide of the involved area on all sides.

Pathological report of the material was made by Dr. James Ewing and is as follows:

"Specimen is a mass of breast tissue 5 x 8 cm. It consists of solid opaque, finely lobulate opaque, reddish-yellow tissue without cicatricial char-

acter. At one point there are several firm nodules which are ducts distended with inspissated yellow material. No definite sign of carcinoma. The ducts are widely dilated and filled with inspissated exudate fatty material and structures quite typical of miliary tubercles. No signs of carcinoma."

There has been no untoward development since that time, the patient's general health has remained excellent, the breast has remained soft without any evidence of disease and the patient has gained 15 or 20 pounds in weight.

The case was presented, not because of the comparative infrequency of tuberculosis of the breast, but to call the attention to the possibility of treating some of the cases, with limited invasion of the breast, by local excision, rather than mastectomy.

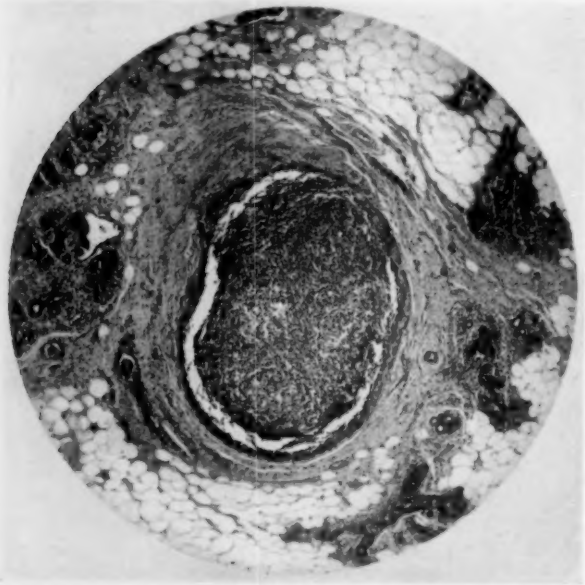

FIG. 2.—Tuberculosis of breast. (Low power.)

DOCTOR LILIENTHAL said that he thought this must be a rare case as all his patients, and those of whom he had previously heard, had pulmonary tuberculosis.

DOCTOR COLEY said that he had observed three cases of tuberculosis of the breast, two of which he had operated upon. One of these cases was a woman, aged twenty years, from whom he had removed the diseased portion without removal of the entire breast. The patient made a good recovery and is still in good condition twenty years later. In this case there was no involvement of the lungs.

DR. WALTON MARTIN said that he had always considered it wiser, in these cases, to remove the whole breast because one surely in that way goes wide of the diseased tissue. In this case, however, the focus was small and Doctor Lee's procedure has proven to be very satisfactory.

DR. JOHN A. MCCREERY said that he considered Doctor Lee was fortunate in finding the lesion to be so circumscribed. In the few cases he had himself seen the disease was so widely disseminated that removal of the entire breast was necessary.

RECURRENT NEUROGENIC SARCOMA OF THE BREAST

DR. BURTON J. LEE presented a woman, sixty years of age, who entered the Memorial Hospital, November 10, 1926. Six years before, she had first noticed a small mass in the left breast. Five years ago an amputation of the left breast was performed at St. Catherine's Hospital in Brooklyn, and she

had remained in good health since then, with no evidence of disease, until about six months ago, when she first noticed a small lump beneath the operative scar. This mass had greatly increased in size up to the time of admission. There had been no pain at any time and no cough. The patient thought that she had lost a slight amount of weight in the few months prior to admission.

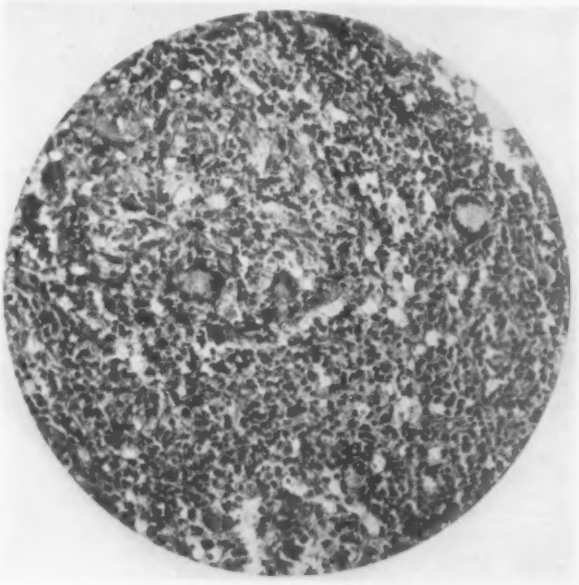

FIG. 3.—Tuberculosis of breast. (High power.)

Physical examination at the time of admission showed a woman of middle age, rather short and stoutly built, who seemed in good general health. There was a vertical scar over the left anterior chest region about twelve centimetres long. Underlying the lower half of this scar was a prominent tumor, 8 x 8 x 10 centimetres, projecting forward. This mass showed considerable fixation to the overlying skin and was of a hard consistence, with indefinite nodulation over its surface. The mass, itself, was fairly movable upon the deeper structures of the chest wall. There were no nodes palpable in either axilla, and although there was slight fulness in the left supraclavicular region, no nodes could be palpated here.

FIG. 4.—Recurrent neurosarcoma of the breast. Photograph before operation.

Röntgenographic examination of the chest, made by Doctor Herendeen, was reported as follows:

"There are adhesions of the right lung to the diaphragm. The heart and superior mediastinum appear normal. The large left breast throws a dense shadow, obscuring the lower half of the left lung. The diaphragm on this side can be seen, however, and appears normal. No definite evidence of pulmonary metastasis."

Physical examination of the chest revealed no abnormal signs.

Two pre-operative low voltage X-ray treatments were given over the region of the tumor and the adjacent portions of the chest wall. The first was given

November 4, 1926, and the second November 6, 1926, with the following factors: Time, 25 minutes, 4 m. a., 10-inch spark gap, 5-mm. aluminum filter, 15-inch focal distance. One treatment was applied anteriorly and the other laterally.

FIG. 5.—Neurosarcoma of breast. (Gross specimen.)

November 11, 1926, under a general anæsthetic, the tumor was excised by Doctor Treves through a transverse, elliptical incision, going wide of the tumor and extending well out toward the axilla, removing all structures down to the chest wall.

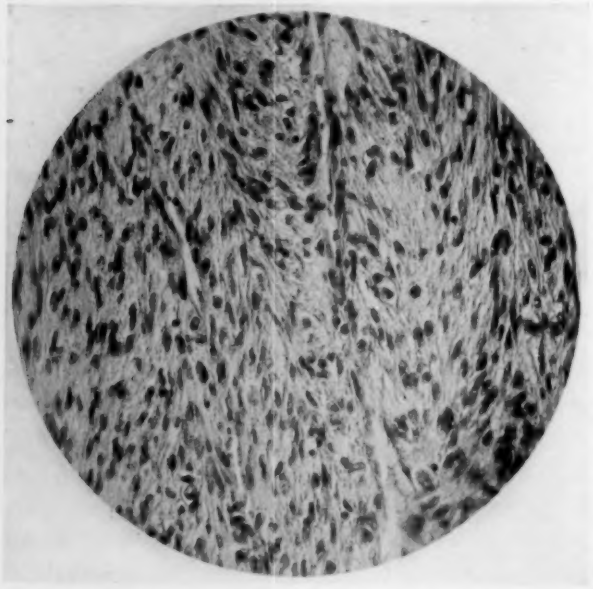

FIG. 6.—Neurosarcoma of breast. (High power.)

Pathological report upon the material was made by Dr. James Ewing and was as follows:

"The breast is the seat of a single solid circumscribed encapsulated tumor mass 7 cm. in diameter. It is very firm, opaque on the periphery œdematous softer, with one necrotic spot in centre. Tumor has the appearance of a fibrosarcoma. Spindle-cell sarcoma fasiculated, somewhat like a neurosarcoma, no epithelial elements."

There was a slight spreading of the wound, due to too early removal of the sutures, but the area of separation healed in rapidly and had practically closed at the time the case was presented. The case was presented for the following reasons:

First: Because of the great infrequency of this type of neoplasm in the breast.

Second: Because most of the neurosarcomata operated upon recur quite early.

Third: Because it seemed desirable to call the attention of the Society to this type of malignant neoplasm, which, in other parts of the body, is sometimes not recognized.

PHLEGMONOUS GASTRITIS

Dr. John C. A. Gerster read a paper with the above title, for which see May Annals of Surgery, vol. lxxxv.

Dr. H. H. M. Lyle said that twenty years ago when working on the etiology of linitis plastica he tried to trace the autopsy records of some cases in which phlegmonous gastritis had been given as a possible cause of linitis plastica so he could appreciate the amount of hard work Doctor Gerster had put on this paper.

As to the suggestion made by Doctor Gerster that some of the an-acid stomach cases which die after operation may have a localized form of this lesion starting from the gastric operative wound, Doctor Lyle had in mind one such case, and he believes that if routine autopsies were done on all cases one might find it a more common lesion than had been thought.

Undoubtedly more cases of acute phlegmonous gastritis occurred after inhalation or ingestion of the irritating war gases than are recorded. Doctor Lyle had personally seen cases where the gastric mucous membrane had been so destroyed. One of the reasons why more cases were not reported was that only a small portion of the gas cases were autopsied and a still smaller portion had a complete autopsy. Very often the autopsy was quickly done in order to find out what kind of a gas was used and not infrequently only the lungs, heart and liver were examined. It is known that many of the vesicant gases caused marked infiltration and hemorrhages into the submucosa with a resulting destruction of the overlying mucous membrane.

Dr. Kirby Dwight reported a case of phlegmonous gastritis on the Medical Service of Doctor Floyde at Roosevelt Hospital. The patient was a man, fifty-four years of age, who came to the hospital last July with dysphagia as the principal symptom. A diagnosis of carcinoma of the œsophagus was made and verified by X-ray and œsophagoscopy. He was in fair condition. Physical examination showed very little; a small lymph-node in the left supraclavicular fossa and a mass in the abdomen above the umbilicus and below the liver, the liver projecting below the costal margin were the only abnormalities. Temperature was normal. White blood-cells numbered 11,000, polymorphonuclears 66 per cent. Free HCl 0; total acidity, 9. He improved under a well-balanced diet and was discharged after three weeks' stay in the hospital and remained well for four months. In November he developed an acute condition and began to vomit, was alarmed and returned to the hospital. He vomited repeatedly after that and had severe epigastric pains, the temperature ran from 101° to 104°, being more often at the latter figure. He died on the sixth day after admission. On opening the abdomen at autopsy the viscera were examined *in situ* and the hardened pyloric extremity of the stomach was palpable below the free margin of the liver. The entire wall of the stomach was indurated and pus came from it on

exploring it with a needle. Microscopic examination showed acute inflammation with miliary abscesses.

As to the possibility of linitis plastica following cases of phlegmonous gastritis, this depends on the definition of linitis plastica. In the old days many cases were reported to be linitis plastica which would not be so considered now. In that time there was a distinction made between leather-bottle stomach and linitis plastica, and the latter was considered to be benign and many kinds of benign processes were classified as linitis plastica. It would seem now, if one follows the teaching of present-day pathology, that it could only be an apparent linitis which could follow an acute phlegmonous gastritis.

Doctor Lilienthal asked Doctor Gerster if he thought that certain cases of supposed tumor formation, which were never diagnosed and which were apparently cured by gastro-enterostomy, might be of this type. For instance, in one of his cases there was a large pyloric mass with symptoms of obstruction which he had felt sure was a tumor and in which he did a gastro-enterostomy as the first of a two-stage operation; and when he went in two weeks later to do a resection he found a perfectly normal pylorus. He had always thought this was a case of cellulitis of the stomach, but it might have been what Doctor Gerster described as chronic phlegmonous gastritis, an infiltration with or without pus between the musculature and the submucosa.

Dr. Walton Martin said that Doctor Gerster had grouped together all the infections of the stomach wall. It is well known that there are a number of chronic infections such as syphilis and tuberculosis as well as the acute infections. This entire group had been considered together, including linitis plastica. It is a difficult question to take up in that way. The acute, diffuse phlegmon is one variety; the more subacute localized infection is another. These form two distinctly different lesions due to pyogenic micro-organisms. It is an interesting question to consider where the organisms come from in the acute diffuse phlegmon. Is the starting point a bacterial embolus or is there a break in the mucous membrane? Doctor Gerster has classed together a heterogeneous group of very rare lesions and has brought out a number of most interesting points concerning them.

Doctor Gerster, in closing, said it was common to get a history of previous attacks, lasting a week or two, which had occurred several months to a year before the final one. In two of the ten cases of resection which ended fatally, the patient made a good operative recovery, but died within six weeks after the operation. In one case death was due probably to acute perforation, in the other to a fatal hemorrhage from a ruptured varix of the splenic vein.

Regarding the suggestion that linitis plastica is a preliminary stage of phlegmonous gastritis, this was merely offered as a tentative surmise.

A number of the patients suffering from chronic phlegmon were afebrile at the time of operation, and the condition was, therefore, mistaken for

carcinoma until the resected specimens were subjected to pathological examination.

The speaker had had experience similar to that of Doctor Lilienthal in performing a gastro-enterostomy for a large movable pyloric tumor causing stenosis with gastric dilatation and grossly resembling carcinoma, which at secondary operation a few weeks later was found to have entirely disappeared, from which it was evident that the tumor must have been of inflammatory character.

Regarding post-operative phlegmonous gastritis, when one considered the clinical course and gross pathological findings in fatal cases there appeared to be several instances in which the likelihood of post-operative phlegmonous gastritis might reasonably be assumed, but in the absence of microscopical examination conclusive proof was lacking.

Doctor Gerster said that Doctor Archibald Malloch had furnished him with the history of still another case as follows:

Walter J., fifty-eight years, admitted to Royal Victoria Hospital, Montreal (service of Dr. George A. Armstrong), March 28, 1914; died March 29, 1914.

Six days before admission there was sudden onset of epigastric pain, which gradually increased in severity, vomiting, the vomitus being chiefly bile, and chills.

Physical examination on admission showed a moribund man, with moderate abdominal distention; epigastrium was somewhat resistant. Temperature ranged between 102° and 107°. Pulse was imperceptible; respiration 36. White blood-cells, 9600.

Diagnosis: Acute pancreatitis or perforated ulcer.

The patient died nine hours after admission. Autopsy showed no peritonitis; diffuse phlegmonous gastritis from pylorus to cardia, confined to submucosa; no ulcer.

Cultures gave streptococci.

Microscopical examination showed œdema of mucous membrane. The submucosa was thickened to five or six times normal width, and was crowded with pus cells and streptococci. The muscularis was involved to the peritoneal coat. The duodenal mucous membrane was entirely normal.

BRIEF COMMUNICATIONS

RUPTURED GASTRIC ULCER IN INFANCY

As a result of the rarity of ruptured ulcer of the stomach in children little interest has been manifest in the disease. A study begun with the idea of obtaining rather than imparting knowledge upon the subject, revealed that the literature dealt mainly with reported clinical cures of cases which have not undergone perforation, or with autopsy reports of few very young infants in whom either death occurred from perforation or hemorrhage.

NUZUM [1] reports two cases, one in an infant twenty-four hours old who vomited two ounces of blood upon three occasions and the conclusion was drawn that she had a gastric ulcer which had been present before birth. The other case was that of an infant fourteen hours old who died, and the autopsy revealed a perforated ulcer of the duodenum.

BISSET [1] reports a case of death from hemorrhage in an infant forty-five hours old. Autopsy showed an acute gastric ulcer with clear cut perforated margins situated on the posterior wall of the stomach near the cardiac opening.

FINKELSTEIN [1] of Berlin, states that duodenal ulcer is rarely seen in the newborn, and reports only five cases where an ulcer of the duodenum was found in the first few weeks of life.

An infant six months of age who vomited large quantities of coagulated blood and passed tarry stools, was reported by Shannon [2] as a case of gastric ulcer. The child subsequently died of pneumonia a few months later, but no autopsy was performed.

KOEHLER [3] is of the opinion that ulcers of the stomach in children are not very common and describes in detail the history of a school-girl aged eleven years, who eventually was operated upon for multiple peptic ulcers. Her recovery was uneventful and several months after the operation was well and strong.

LOUDON [4] records an interesting history of a child two months old suffering from duodenal ulcer. The baby died from hemorrhage and a post-mortem examination revealed an acute ulcer on the posterior wall of the first part of the duodenum, while a second small ulcer was situated near the pylorus on the anterior wall of the duodenum.

A number of different theories have been advanced as to the cause of these ulcers. Hunter is of the opinion that infection in childhood may cause it, and Weber also believes that preceding diseases are a predisposing factor. Roseman and Anderson found structures resembling stomach ulcer in guinea pigs that had died of diphtheria. Loudon is inclined to the belief that thrombosis of the umbilical vein may cause thrombi in the vessels of the duodenum which probably lead to ulcer. Weber [5] supports his views by describing two cases of gastric ulcer which began in the eleventh and ninth years of life, respectively. Both children were operated upon and made uneventful recoveries.

A striking feature of acute ulcer, according to Alden,[6] is the large percentage of perforations. The first evidence of the presence of ulcer may be a sudden hemorrhage or pain from perforation, although the symptoms may be masked by those of an infectious disease. Pain and tenderness was noted by Alden in 13 per cent. of the acute cases, and in over 60 per cent. of the chronic cases. It was localized in the epigastrium in older children and in

younger children it seemed to be over the entire abdomen. Vomiting usually followed diarrhœa in acute cases in infants. In older children it followed epigastric pain and relieved the diarrhœa. Hæmatemesis was noted in 40 per cent. of the cases. Fever was noted in a great number of acute cases, at times as high as 106 degrees. Diarrhœa was a prominent symptom and was present in almost half the acute cases.

My personal knowledge of the subject is confined to one case and can be neither very definite or convincing, but the case presents some features which in the light of the operative findings seem worthy of record.

Report of Case.—S. H., a girl, fourteen months of age, a patient of Dr. Ralph Bell of Media, Pa., was admitted to the Chester Hospital, June 6, 1926. The child was examined by Dr. Elizabeth Clark, to whom I am indebted for the following history: One week ago the child became restless and feverish. She vomited several times and evidently had abdominal pain. An obstinate constipation had become a marked feature of her condition. She was a full time baby. Normal delivery, breast fed until nine months old. Always healthy until three months ago when she contracted measles and this was followed, two months ago, by an infected finger from which pus freely discharged.

The patient was a well developed baby fourteen months old. Very restless and continually rolling its head. The skin is dry and hot. Not emaciated but some evidence of dehydration is present. Temperature 102, pulse 140, respiration 60. A mass appears below umbilicus, its longest diameter transverse, definitely outlined and tender to palpation. The abdomen is not rigid. In upper abdomen and in flanks intestinal patterns are apparent. A post-operative note which I made was appended to the history: "Could not definitely make out the mass described above. It impresses me more as a rigidity of the recti muscles. The rigidity is not board-like in character and there is no tenderness above the umbilicus. The point of greatest tenderness is just below the umbilicus in the midline.

Operation.—The peritoneal cavity was opened by a right rectus incision mostly above the umbilicus. As soon as the parietal peritoneum was incised, turbid yellowish fluid began to exude from all portions of the peritoneal cavity. The intestines were distended and inflamed. There was no invagination present. Appendix not inflamed. The incision was extended upward and the stomach explored. A perforated ulcer, large enough to admit the little finger, was found on the lesser curvature 3 cm. proximal to the pylorus. The edges were inflamed and friable. The perforation was closed by four interrupted sutures and the gastro-hepatic omentum was stitched down over the wound. The pelvis was drained with a rubber tube and the abdominal incision closed in the conventional manner. The child never reacted from the operation and died in about four hours.

Summary.—Ulcer of the stomach or duodenum is uncommon in children, but should be considered as a possibility in infants when collapse follows a profuse hemorrhage from the stomach or bowels. A ruptured ulcer should be suspected when severe abdominal pain, tenderness and constipation persist without the localizing symptoms of the ordinary causes of a surgical abdomen in infants. Particularly should we suspect ruptured ulcer if added to these symptoms there is present a history of preceding infection or infectious disease.

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TUBERCULOSIS OF THE BREAST

In view of the numerous articles appearing during the past year with tuberculosis of the breast as their subject, it may be worth while to report the following case. It seems to be a definite example of spread of the disease from the axillary lymph-glands to the breast.

A married Chinese woman of thirty-one, entered the hospital October 12, 1926, complaining of a swollen and ulcerated right breast. One year previously she had noticed a small pustule in the right axilla. At that time there was some redness and induration of the surrounding tissue. There was no pain. A short time after this a similar nodule appeared somewhat nearer the breast, and two months before admission these nodules had broken down and the contiguous breast tissue had become involved. From that time on the breast became more and more extensively involved, with the formation of a large ulcerated area with much purulent discharge, redness, induration, and pain of the rest of the breast. There were daily fever and loss of appetite and strength. During the greater part of her illness she had been pregnant, and on admission was nursing her seven-day-old baby, using the left breast. Except for aching pain in her right arm, and headache, she had no other symptoms, and had never before been sick. She was a fairly sick-looking woman, with the positive findings limited to the right axilla and breast. The left breast was a normal lactating one. The right breast was diffusely enlarged, and there was a large sharply margined ulcer, occupying about half of the upper quadrant of the breast. Just medial to this was a similar but smaller one separated from it only by a band of skin. Its base was dirty and granulating, and there was a profuse purulent discharge. The surrounding skin was blue-red in color. There were two similar but smaller ulcers situated in the axillary prolongation of the breast. A mass of glands could be felt high in the axilla. There was no evidence of tuberculosis elsewhere in the body.

Laboratory Findings.—Hæmoglobin, 70 per cent.; red blood-cells, 3,464,000; white blood-cells, 9200, with 93 per cent. polymorphonuclears and 7 per cent. mononuclears. Urine showed a slight trace of albumin, a trace of sugar and a few pus and epithelial cells. Smear from the ulcer showed no tubercle bacilli.

With a clinical diagnosis of tuberculosis of the breast and axillary glands, operation was done October 23, 1926. The ulcers were carefully cleaned and then treated with pure carbolic acid, and gauze was then sutured over these areas. The breast and the adjacent involved axillary tissue was excised without disturbing the pectoral muscles. In spite of a careful attempt to keep wide of the involved tissue, the process had so undermined the skin that it was encountered in several places. The axilla was found to contain two large groups of glands which were removed. The wound was left open and dressed with gauze soaked in saline solution. Rectal ether anæsthesia was used.

The immediate post-operative course was stormy. The wound did not clear up sufficiently for skin graft until December 9. This was done by transferring small deep grafts from the thigh. The grafts did well except at the margins which continued a

low grade infection until her discharge December 28. The axilla was especially sluggish in clearing.

Pathological Report.—The specimen consists of a breast and two masses of lymph-glands. The surface of the breast presents a large ulcer measuring 2 inches by 1½ inches with sharply defined margins and a blue-red discoloration of the immediately surrounding skin. Two smaller ulcers are present in the flap of tissue attached to the breast. The base of the ulcer is made up of dirty pale granulation tissue. On section the granulation tissue forming the base of the ulcer is seen to extend down for a depth of 1½ inches. There is some supporting fibrous tissue, but no glandular tissue is seen. The bases of the other ulcers are similar. Several of the glands are caseous. Sections were taken from the base of the main ulcer and one gland. These sections were studied at the Peking Union Medical College Hospital, and the following report made:

Microscopic Examination.—The section is composed mainly of homogeneous, pink-staining, necrotic tissue and very poorly preserved granulation tissue showing a large number of giant cells with peripherally arranged nuclei. Within this granulation tissue are pockets completely filled with polymorphonuclear leucocytes and cellular débris. There is extensive infiltration throughout the section with large mononuclear phagocytic cells, lymphocytes and large numbers of polymorphonuclear neutrophilic leucocytes. In two areas the tissue is almost completely composed of lymphocytes which are arranged around vessels containing blood and coagulated fluid; although one suspects that these may be the remains of lymph-glands, no peripheral sinus or other structure remains by which they can be identified. Another section shows, in addition to these changes, a large branching duct lined by several layers of rather poorly preserved columna epithelium. It contains cellular débris and numerous polymorphonuclear leucocytes, and is surrounded by loose connective tissue, which is densely infiltrated with wandering cells and lymphocytes. In another portion of the section definite small islands of mammary glands can be made out. About these is a dense infiltration of wandering cells, several of them being practically replaced by poorly preserved granulation tissue and large giant cells. Within this granulation tissue a number of well-formed tubercles are seen. Diagnosis: Tuberculosis of breast with secondary acute and chronic infection.

It would seem that the history of spread of the disease from the axilla, the finding of definitely caseous axillary lymph-glands, and the tuberculosis of the breast would justify the assumption that this case started in the axillary lymphoid tissue.

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ANEURISM OF THE INTERNAL CAROTID ARTERY*

Extra-cranial aneurism of the internal carotid artery has been reported one hundred and six times. With early recognition and proper operative procedures, the majority of the patients should recover; but in the past a large number of the patients have died because of failure of recognition, blundering or delayed treatment of the cases. Treatment by operation on the carotid vessels offers the best prospect of relief, and in view of the findings in the one hundred and six reported cases, it should be relied on in caring for practically all of these cases.

Winslow [1] has presented a full analysis of these cases with clear clinical observations and conclusions that should receive wide acceptance.

* From the service of Dr. V. P. Blair, Washington University, St. Louis, Mo.

The following report is presented to continue the work of Winslow in calling attention to the condition.

Woman, thirty-six. Family history and past history generally negative. No history of syphilis (Wassermann negative). Married. Two children, living and well.

P. I.: A swelling appeared under jaw in 1919 without symptoms, but very slowly enlarged.

In October and November, 1923, there were two short periods of numbness of the right hand and loss of holding power.

In December, 1923, she was suddenly paralyzed over the right side, could not talk for three hours, but did not lose consciousness. In bed one day.

In June, 1924, upper teeth were pulled.

In December, 1924, the tonsils were removed and a cyst of some sort is said to have been removed from the mouth at the same time.

In February, 1925, she had another stroke similar to the first one. In bed one day again. This time her face was partly paralyzed.

In April, 1925, the third and last stroke occurred. In bed two weeks and has been weak on the right side ever since. When admitted to the hospital in June, 1926, she stated that her general condition had been getting better since April, 1926, but the tumor in her neck was enlarging and had been doing so since the tonsillectomy in December, 1924. She had been having pain in the jaw and side of head while eating, some frontal headache, some nose bleed, black spots before eyes, and some impairment of vision of the left eye. Chief complaint was difficulty in swallowing and pain.

She was a thin, worried looking woman, able to be up and around; no apparent disturbance of gait. There was a smooth, rounded swelling below and forward of left angle of jaw about 5 x 4 cm. in size; pulsation and bruit present. Floor of mouth raised and tongue pushed to right by the swelling. Few palpable glands in neck. Pupils R > L. E. O. M. normal. KK hyperactive on right with questionable Babinski. General weakness on right. Blood-pressure, 100/80.

This patient had had on the four previous occasions some cerebral anæmia, probably due to a blockage of the blood-flow through the aneurismal sac. It was thought that these impairments of cerebral circulation might have already accomplished the same precaution that fractional ligation or preliminary occlusion of the common carotid would, and immediate occlusion of the common carotid was decided on if it should prove necessary at operation.

Operation, June, 1926, by Dr. V. P. Blair. Left Kocher incision, with a second one down along sternomastoid. Glands large, translucent and friable; specimen removed for diagnosis. Left common and external carotid, superior thyroid, lingual and facial arteries ligated. Main mass just under ramus exposed and opened and 30–40 c.c. of thick, dark, old blood escaped. Not much further bleeding. Cavity and wound packed with iodoform and Balsam of Peru gauze.

Uneventful post-operative course with patient up and about soon afterwards and trying to do her housework, but too weak for much of it.

December, 1926. Reported some nausea and faintness at times; was about four months pregnant. KK, biceps and triceps R > L. No clonus. Pupils round and equal, react to L and A. E. O. M. normal. Wound entirely healed. Patient appeared cured.

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J. B. Brown, M.D.,
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BOOK REVIEWS

ATLAS OF THE HISTORY OF MEDICINE—ANATOMY. By DR. J. G. DELINT, Lecturer on History of Medicine at the University of Leiden. With foreword by Charles Singer. Paul B. Hoeber, New York, 1926. Cloth, folio, pages 96.

A series of figures illustrating the history of anatomy; a few plates illustrative of the crude anatomical ideas of the Dark Ages up to the time of Leonardo DaVinci and Vesalius illustrate most strikingly the real creation of anatomical illustrations which those two geniuses accomplished.

The main interest and value of the book, however, is in the portraits of the Masters of Anatomy which constitute the greater part of the book. Of course, Hippocrates, and Aristotle and Galen and Celsus are included and the dissection scene from DeKetham, DeChauliac and Mordino are not omitted but the real interest begins with Guinther of Andernach, the Master of Vesalius and of Vesalius himself followed by Fallopius, Ingrassias, Colombo, Coiter, Fabricius of Aquapendente, Severinus, Bauhinus, Paauw, Valverde, Servetus and Harvey, Riolan and Hoffman and divers other seventeenth century anatomists, every one of which the student recognizes as associated with particular parts of the human anatomy which bear their names, down to Cheselden, the Hunters, the Bells of later date, and finally Huxley, Schwann, Haeckel and Darwin of the last generation.

The writer can imagine how much pleasure and interest such a book would have given him when, as a student, he was becoming interested in the problems of human anatomy. It is given to but few to enjoy the possession of the original books which preserve to the world the work of these masters. Such a book as the present one, which may properly be called an index to the work of the Great Masters, fills a valuable place and ought to be attractive to every scholarly medical student or practitioner of the present day. The editor of this volume expresses its aim in these words: "A classical picture of an anatomical procedure, a portrait of one of the heroes of anatomy, or the title page of one of the great anatomical treatises of the past can awaken the memory of some anatomical discovery which may become fixed thereby. To aid the study of Anatomy in this way is the chief aim of this atlas. But its author would ask for it also a place in the library of the practitioner who may desire to occupy his leisure with the study of the History of Medicine."

The book certainly fulfills the object aimed at by its compiler.

LEWIS S. PILCHER.

INFECTIONS OF THE HAND. By LIONEL R. FIFIELD, F.R.C.S., Surgical Registrar, London Hospital. Paul B. Hoeber, New York, 1927. Cloth; 12 mo; pages 192.

This is a hand-book that one may slip readily into one's overcoat pocket. It has, however, treated its subject in a very complete and satisfactory manner

the more so when are taken into consideration the peculiar difficulties of the subject incident to the intricate anatomy of the region involved, and the frequency and importance of the infections which so often result in irreparable damage to important structures and functions.

It is a simpler book than the masterly monograph of Kanavel. Every attempt is evidently made to keep the size of the book within moderate limits and the wonder is that in so condensed a manner, the author has been able so clearly to present the important essentials of the subject. It is not only profusely but also intelligently illustrated. The cuts show dissections, serial cross-sections, injections of synovial and cellular tissue spaces and many radiographs.

Naturally, the subject belongs rather to the domain of minor surgery and will appeal more especially to the advanced student and to the general practitioner. It, at the same time, however, is worthy the occasional attention of the advanced surgeon. We commend the book to the attention of every one who may have to do with a hand infection.

LEWIS S. PILCHER.

SOUTH AMERICA, AMPLIFIED TO INCLUDE ALL OF LATIN AMERICA. By FRANKLIN H. MARTIN, Director General American College of Surgeons. Revised edition. 12mo, cloth, 435 pages. Fleming H. Revell Co., N. Y., 1927.

It was the writer's privilege to spend the winter of 1923 in South America. The picturesque scenes of Rio de Janeiro and Buenos Aires gave an excellent introduction to a two weeks' sojourn on the shore of Lake Nauhel Huapi in Patagonia and thence across the Andes by boat and mule back to Chile at the southern end of the Chilean Central Railway, then up the great central valley of Chile to Santiago and thence to Valparaiso and Lima and Tacna and Panama, all of which experiences were the subject of a chapter in our "Surgical Pilgrim's Progress." During the whole journey, however, we carried with us the original hand-book of Doctors Martin and Mayo which had been published only the year before. We found the book a mine of information and a most valuable guide for travel, more particularly so for the medical man, inasmuch as the spirit which had prompted its compilation had been to promote a better acquaintance between the physicians of North and South America.

Now here, four years later, comes a second edition, revised and amplified by additional chapters which include all of the Latin American countries. The surgeons and medical institutions are dealt with extensively. The general status of the medical and surgical profession in that part of the world should be of special interest to us for the frequency with which the professional men of the North American states are being drawn to visit South America and the Isthmus in their vacation trips is increasing and is likely to become more

marked as the years go by. From the point of picturesqueness and general interest, the trip equals that to any other part of the world. From the point of restfulness, it is ideal.

LEWIS S. PILCHER.

HUMAN PATHOLOGY. A Text-book. By HOWARD T. KARSNER, M.D., Professor of Pathology, School of Medicine, Western Reserve University Cleveland, Ohio. With an introduction by Simon Flexner, M.D. Philadelphia and London, J. B. Lippincott Company. 8vo. Cloth, pp. 980.

THE object of this book is to present the morphological alterations incident to disease, in the light of modern views as regards their functional significance. In view of the fact that this is a text-book and because in the study of pathology an acquaintance with the names of the pioneers and the outstanding figures in this subject is desirable, a list of such names is found at the end of each chapter. The references are so complete that in several instances there are upward of 100 names given, along with the titles of the monographs and the volume, page, date and name of medical journal in which they appeared. To discuss the text content is unnecessary in a review of this sort. Suffice it to say that every conceivable pathological process to which human flesh may fall heir is fully, completely and intensively discussed, the author keeping in mind constantly its relation to clinical and functional disturbances.

The introduction written by Simon Flexner abounds in sincere praise for the author. In conclusion Doctor Flexner states that this text-book presents the subject of pathology as now conceived and taught in this country and in Europe in a manner suitable for the medical and biological student as well as for the practitioner of medicine desiring to keep abreast of this ever enlarging subject. He states further that the prospective reader is to be congratulated on having available such a text-book as is this.

MERRILL N. FOOTE.

UROLOGY. A Text-book. By OSWALD SWINNEY LOWSLEY, M.D., of the Brady Foundation of the New York Hospital, and THOMAS JOSEPH KIRWIN, M.D. 8vo, cloth, pp. 699. Lea and Febiger, Philadelphia, Pa., 1926.

THIS single volume of 699 pages consists in a complete résumé in the form of a text-book of Urology. It is based upon eighteen years of practical and intensive study and clinical observation. As a background for the work the authors have used the methods of the old masters of the subject picking out the procedures which time has proven to be reliable and efficacious. To this background they have added the modern modifications which have brought the work up to date. The elimination of the non-essentials has been such that the book contains only procedures of value.

The anatomy, embryology, morphology and anomalies are given due consideration. The operative technic is well illustrated by numerous plates and diagrams of which there are 246. Of especial interest is the portion which deals with regional anesthesia.

The book is divided into three parts. The first part concerns itself with an introduction along with the complete but condensed history of urological surgery beginning with the ancient Hindoo literature and coming up to the present day urologists. The second part deals with diagnostic procedures in urology. The third part in which there are ten chapters deals with the diseases of the genito-urinary organs and their treatment.

The book is so condensed yet complete, the illustrations are so vivid yet not too diagrammatic; its presentation is so detailed and individual yet so straightforward and easily comprehended that it is highly recommended for not only the urological surgeon but also for the use of the general surgeon and practitioner of medicine.

MERRILL N. FOOTE.

CHANGE OF EDITORIAL ADDRESS

The office of the Editor of the Annals of Surgery has been changed to 489 Washington Avenue, Brooklyn, New York. All contributions for publication, Books for Review, and Exchanges should be sent to this address.

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